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THE SURGICAL TREATMENT OF HERNIA*

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The surgical treatment of hernia is an important subject as no present day technic entirely obviates recurrences. The aim of every surgeon should be to make such recurrences as few as possible. Recent improvements in technic have made the present review of the status of the surgical treatment of hernia advisable.

In this paper three aspects of the subjects will be considered.

I. RECURRENT HERNIA AND THE USE OF FASCIAL GRAFTS

The surgical treatment of recurrent hernia, especially in so far as it pertains to groin hernias (indirect inguinal, direct inguinal, and femoral hernias) involves the original procedure as well as the secondary repair. In so far as the original procedure is concerned, I am of the belief that non-absorbable sutures are important in reducing the incidence of recurrence. Furthermore, each step of the original repair should be done carefully as is outlined in Section III of this paper. The rule of Fallis gives a significant lead as to the causation of recurrence of groin hernias. This rule is that hernial recurrences following operations by occasional surgeons establish themselves as indirect recurrences, whereas hernial recurrences following operations in large surgical centers establish themselves as direct recurrences. The interpretation of this rule is that every hernia tends to recur as a direct recurrence, but when the operation is improperly done the sac is often tied off too low and the additional factor of an indirect recurrence exists. The author has personally used free fascial grafts in the repair of eleven recurrent groin hernias and has used interrupted silk sutures with a Cooper's ligament technic in a much larger series of cases. The conclusions, as outlined in a paper by Swenson and Harkins (1943) are that silk is just as good as fascia in most recurrent groin hernias.

II. LARGE VENTRAL AND INCISIONAL HERNIA AND THE USE OF CUTIS GRAFTS

In the repair of large ventral and incisional hernias the important element is to free the fascial ring which, incidentally, is often much smaller than it would first appear to be, and then to suture the edges of this fascial ring to each other. The author has personally never seen an incisional hernia where the fascial edges could not be brought together. Fascial and other types of patches should be used, therefore, only as an adjunct to the major repair and should not be applied to bridge a defect. In performing the major repair free fascial grafts and non-absorbable sutures are, in my opinion, the agents of choice and, of these, non-absorbable sutures are preferable. In most instances, a large hernia will have to be repaired longitudinally, since the original abdominal incisions are in most instances longitudinal. In some cases, however, vertical flaps may be used. Practically always some overlapping of the

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flaps utilizing mattress sutures by a modified Mayo technic is feasible.

In eleven cases (1945) I have used cutis grafts as a patch over the larger incisional hernia repair. In all of these instances silk was used to draw the fascial flaps together and the cutis graft was sewed down over the suture line. Cutis is the deep layer of the skin and it is in general prepared by removing the epidermal layer, preferably with a Padgett dermatome, at a depth of about 10/1000 of an inch. The deep layer of the skin is then removed and used for the cutis grafting. The epidermal piece is then sutured down over the donor defect. A piece of cutis about 3x5 inches was used in most of the clinical cases. Microscopic sections revealed that no matter how thick a layer of epidermis was removed, some epidermal elements still remained in the cutis grafts. In dog experiments these lead to sebaceous cyst formation, but in the eleven human patients no difficulty was encountered and so far all of the repairs have remained intact.

III. GROIN HERNIAS AND THE USE OF A COOPER'S LIGAMENT REPAIR

Most groin hernial repairs involve the suturing of the conjoined tendon to Poupart's ligament. The technic I advise involves the suturing of the conjoined tendon to Cooper's ligament. This operation was originated by Lotheissen in 1898 and was shown by McVay (1941) to rest on a sound anatomic basis. This operation was reported by Harkins, Szilagyi, Brush and Williams (1942) as done on a series of 131 groin hernias, and in 1943 by Harkins and Swenson in a larger series of cases. At the present time this series adds up to 362 groin hernias treated by the Cooper's ligament technic and while the follow up is far from complete, I know of only one recurrence. Recently this method has been utilized by Mattson (1946).

Essentially the Cooper's ligament repair of groin hernias involves the following ten steps:

1. *Exposure of the cord and opening of the indirect sac:* In all cases, whether the hernia is indirect inguinal, direct inguinal, or femoral, the indirect sac is opened. In many cases of direct inguinal hernia or femoral hernia, the indirect sac will be normal in size, but can always be found superior and medial to the cord. In cases of complete indirect inguinal hernia of the congenital type, or where the hernial sac is long, it is cut across near the internal ring and separated from the cord. The proximal end is closed by an internal purse string suture as outlined in step 4, while the distal end is left in place. Many surgeons are of the belief that leaving the distal sac in place will cause hydrocele. This has not been my experience and I believe that the hematomas that result from removing the distal sac are far more disastrous than any theoretical occurrences of hydroceles.

2. *Exploration:* Once the indirect sac is opened it is a simple matter to insert the gloved finger and feel Hesselbach's triangle for a direct weakness or obvious direct hernia and to feel the femoral ring. It is indeed surprising how few surgeons will take the extra thirty seconds to perform this exploration and many femoral and direct hernias are overlooked by not so exploring.

3. *Hoguèt maneuver:* If a direct sac or weakness is present, or if a femoral hernia is present, it should be transposed laterally to the inferior epigastric vessels by the method of Hoguet (1920). This maneuver is an extremely useful one. In general, no matter how large a direct sac is, it is not opened but is merely transposed. In the case of large direct sacs the transversalis fascia can be infolded with numerous interrupted silk sutures. One advantage of not opening a direct sac is that the danger of opening the bladder is largely obviated.

4. *Internal purse-string closure of the indirect sac:* Such a closure is done with a medium silk suture as high as possible and helps to prevent indirect recurrences.

5. *Plastic on the internal ring:* After the purse-string suture of the indirect sac, it will be noticed that in most cases the internal ring will admit three of four fingers. This should be tightened to give it a

sphincter-like action as advocated by MacGregor (1929, 1945). This tightening is accomplished by suturing the conjoined tendon, the transversalis fascia, and Henle's ligament to each other. The use of Henle's ligament has been especially pointed out by Clark and Hashimoto (1946).

6. *Relaxation of internal oblique:* This is done as directed by Rienhoff (1940) at the junction of the external and internal oblique.

7. *Cooper's ligament sutures:* The conjoined tendon and transversalis fascia are then sutured with heavy silk ligatures to Cooper's ligament. This ligament is an extremely tough thickening on the anterior superior surface of the anterior ramus of the pubis. The first year or so of using this repair I visualized Cooper's ligament by breaking down Hesselbach's triangle. Then for a year or so the sutures were applied blindly, leaving Hesselbach's triangle intact. Recently, however, I have returned again to the principle of direct visualization of Cooper's ligament, having decided that it is easier to apply the sutures under direct vision. The sutures are carried out about one inch to one and one-fourth inches from the anterior pubic spine.

8. *Closure of the external oblique aponeurosis:* In most instances this is done over the cord which is now dropped into the dead space left by the Cooper's ligament sutures.

9. *Closure of Scarpa's fascia.*

10. *Skin closure.*

SUMMARY AND CONCLUSIONS

1. The proper treatment of recurrent hernia involves the choice of a correct original hernial repair as a prophylactic measure and a careful secondary operation as a therapeutic measure. The use of non-absorbable sutures with a Cooper's ligament technic is a satisfactory method for dealing with recurrent groin hernias.

2. The fascial ring should be brought together and closed in ventral and incisional hernias. In certain instances patches of cutis can be grafted over the suture line to give additional support.

3. In a series of 362 groin hernias (indirect inguinal, direct inguinal, and femoral hernias) a Cooper's ligament technic has proved useful.

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PULMONARY HYPERTENSION

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HISTORY

Pulmonary arteriosclerosis was recognized in 1803 by Brisson, but it was thought to be rare until 1815 when Dietrich pointed out its frequency in cases of mitral stenosis. At this time the concept of hypertension of the lesser circulation was a very vague subject and least of all, an entity. From the start of the twentieth century to 1935, secondary cor pulmonale had been attributed to disease of the left side of the heart or to lesions of the parenchyma of the lung, and primary cor pulmonale, to lesions of the vascular bed. Since the work of Brenner in 1935, however, primary pulmonary hypertension has become a disease in which no specific lesion can be found and some

writers even doubt if such a condition really exists. On the other hand, secondary pulmonary hypertension, the clinical curiosity of yesterday, has become the "new disease" of today, with its definite "foot-prints" in the lesser circuit being frequently found at the necropsy table at present.

INTRODUCTION

Although the blood pressure within the pulmonary artery cannot be adequately measured, it is realized that such a hypertension can occur and has become a definite entity in the past few years. It has been confused with "pulmonary congestion", "insufficiency of the right heart", and especially with arteriosclerosis of the pulmonary vessels. This concept explains some of the rarer diseases associated with cyanosis such as Ayerza's disease, some of Geisböck's disease, and some of the cases of the Osler-Vaquez syndrome. The recognition of this type of hypertension is of utmost importance from the point of view of circulatory dynamics, in that the compensatory mechanisms involved play such an important role with regard to circulatory disorders. This paper will be devoted to the circumstances under which pulmonary hypertension occurs, the circulatory dynamics involved, the relation to other circulatory states, and to the clinical recognition of such a condition.

CIRCUMSTANCES ASSOCIATED WITH PULMONARY HYPERTENSION

The immediate cause of pulmonary hypertension is unknown; however, certain circumstances are recognized under which an increase in peripheral resistance arises. At necropsy these cases of increased pulmonary peripheral resistance show, with rare exception, an arteriosclerosis of the larger vessels and also an arteriocapillary fibrosis. As you know, such a condition is found in the greater circulation of the arteriosclerotic person with hypertension. The rare exceptions are the very few cases of decrescent arteriosclerosis found in both circuits in extremely old individuals.

Recently it has been recognized that sclerosis of the pulmonary artery and of the aorta are entirely independent of each other, in fact, the simultaneous appearance of such a condition is the exception rather than the rule. It is also known now that pulmonary arteriosclerosis is entirely independent of age or sex. If some toxin, poison, bacterium, or metabolic product caused arteriosclerosis it seems that the process would have to be one affecting both circuits. The experiments in which high cholesterol diets produced arteriosclerosis seem to produce not a true arteriosclerosis but rather a lipoid transformation of the intimal cells. Jores states that to have true arteriosclerosis one must produce changes in the media and there must be hyperplasia of the elastic fibers. These criteria have not been brought out in the experiments mentioned above. Many of the authorities believe the lesion is probably due to stretching of the tissues resulting in replacement fibrosis with eventual hyalin and calcareous deposition, and compensatory hyperplasia of the elastic fibers, muscularis and intima. The scope of this paper limits any further discussion as to etiology of arteriosclerosis. However, definite conditions are associated with an increase in pulmonary peripheral resistance and these will be elucidated now in their order of frequency:

(a) Mitral disease, either stenosis or insufficiency. This is by far the most common.

(b) Permanent emphysema, whether secondary to asthma or to senility.

(c) Widespread obliteration of lung parenchyma such as fibroid tuberculosis, bronchiectasis, pulmonary abscess, interstitial pneumonitis, carcinomatous lymphangitis, widespread pleural adhesions, or intravascular obliteration of pulmonary circuit.

(d) Cardiac disorders leading to prolonged right heart failure, that is, coronary disease, adherent pericardium, "spent" Graves syndrome, or kyphoscoliosis.

(e) Patent ductus arteriosus or other shunts which run from left to right.

As mentioned before, the underlying factor is an increase in pulmonary vascular resistance and even though one cannot measure this resistance, it can be predicted

that on a purely mechanical basis the pressure in the pulmonary circuit must be raised in these circumstances. Two groups of mechanisms are concerned: one is on an obstructive basis either within or beyond the pulmonary circuit, and the other is arteriovenous shunts in certain cases of congenital heart disease. Mitral disease or a failing left ventricle with its effects proximately are the only examples of obstruction distal to the pulmonary circuit.

A stenotic mitral valve causes an increased tension within the left auricle which is transmitted backward through the pulmonary vein and thence to the alveolar capillaries. Mitral insufficiency likewise increases the tension within the left auricle, but the incidence, however, cannot be as great as in mitral stenosis, for the resistance offered by the systolic discharge of the left auricle against a tight mitral ring must be much greater than against an incompletely emptied left ventricle. Thus the arteriosclerosis associated with mitral stenosis is much more prominent than that seen with mitral insufficiency.

In emphysema, the increased resistance is the result partly of extensive destruction of the capillary bed and partly by compression and stretching of the intra-alveolar arterioles due to the expansion of the alveoli. On the other hand some authorities say that emphysema, *per se* is not an important cause, for although obliteration of the capillaries is extensive, it is extremely unlikely that it ever reaches a degree capable of exhausting the vascular reserve. Also, emphysema is almost always seen in persons who are at an age when myocardial, valvular, coronary and hypertensive heart disease is not at all uncommon. I do not know which "path" to take in respect to this controversy; however, I believe that the increase in pulmonary tension associated with emphysema is exerted equally over the entire vascular circuit and, if true, the amount of vascular reserve would have nothing to do with averting a hypertensive state in the lesser circuit.

Extensive chronic infiltrations of the lung cause an increased peripheral resistance by a destruction of the capillary bed. Pulmonary abscesses with widespread parenchymal reaction or widespread fibroid tuberculosis will greatly encroach upon the vascular bed especially since some arterioles and some of the smaller arteries can be affected by these conditions. Carcinomatous lymphangitis associated with obliterative pulmonary arteritis of small and medium sized arteries secondary to perivascular lymphatic infiltration by carcinomatous cells can very easily destroy enormous areas of the vascular bed. It must not be overlooked that the obliteration of one or two larger radicles of the pulmonary tree will have a more devastating effect than a very widespread encroachment upon the capillary bed alone.

Some writers say that a finding of right sided hypertrophy and dilatation is seen in some patients having sickle-cell anemia. Capillary stasis, owing to distortion and agglutination of erythrocytes, is felt to be the essential factor in producing the obstruction within the pulmonary circuit.

Schistosomiasis (Mansoni) has been shown to cause granulomatous pulmonary lesions within or related to medium and small pulmonary vessels. Many of the small arteries and arterioles showed evidence of intimal thickening and hypertrophy of the media with subsequent encroachment upon the lumen. In many of these patients evidence of right sided hypertrophy and ultimate failure was observed. Most of the organisms ultimately reach the portal system but enough residual infection remains in the lung to cause pulmonary hypertension.

Others state that involvement of the still larger arteries can produce this condition of pulmonary hypertension. In acute cor pulmonale and in thrombosis of the pulmonary artery, if the patient does not die, strain on the right heart can be elicited usually by clinical or electrocardiographic procedures. The authorities are not in accord at all in their beliefs concerning the production of pulmonary hypertension by the last four above conditions mentioned

but I give them to you for further study and thought.

The explanation for the increased resistance in the pulmonary circuit following advanced kyphoscoliosis is not entirely clear. It is partly due to compression of the lungs by atelectasis and compensatory emphysema, to kinking of the pulmonary vessels, and to the attacks of repeated bronchitis to which these patients are subject.

Congenital arteriovenous shunts whereby blood from the left heart is thrown into the lesser circulation will naturally increase the tension in the lesser circulation. When a patent ductus arteriosus is present, the higher pressure in the aorta will cause an extra quota of blood to be thrown into the pulmonary circuit. In most cases of septal defect, the reflux of blood is not enough to cause circulatory embarrassment unless another factor exists which increases the arteriovenous shunt from left to right. An example of this is the combination of mitral stenosis with a patulous foramen ovale. Chronic cor pulmonale associated with the tetralogy of Fallot is due to the pulmonary stenosis rather than to the reflux of arterial blood into the lesser circuit. Acquired arteriovenous shunts can be a cause but its rarity permits little or no discussion on the subject. Watjen reports a case of pulmonary arteriosclerosis in a child aged six months, with transposition of the vessels and a septal defect of the ventricles.

These mechanisms fulfill the requirements "laid-down" for the production of pulmonary hypertension, namely, (1) the minute output of the right ventricle; (2) the resistance and capacity changes in the pulmonary circuit; and (3) back pressure resistance produced in the left heart by changes in the systemic circuit.

CHANGES IN CIRCULATORY DYNAMICS RESULTING FROM PULMONARY HYPERTENSION

Due to interference of the passage of oxygen, because of increased thickness and fibrosis of the capillaries of the alveolar wall, and also with the support given by increased venous pressure and stasis, anoxemia is soon to arise. In shunts between the right and left hearts the anoxia is aggravated by the amount of unsaturated blood that is poured into the left heart. The anoxemia results in an increase in the percentage of circulating reduced hemoglobin and this gives rise to a predominant feature of pulmonary hypertension, namely, cyanosis. When failure of the right heart ensues, the cyanosis is enhanced because slowing of the peripheral capillary flow results in an increase of reduced hemoglobin and the increase in venous pressure as mentioned below. The first compensatory result of anoxemia is polycythemia, produced by hyperplasia of the bone marrow. The succeeding compensatory mechanism is an increase in the circulatory blood volume. This is seen only in long standing cases after repeated attacks of failure. The mechanism is allied to the polyuria that follows progressive renal insufficiency.

Increased pressure in the pulmonary circuit may cause an increase of effective venous pressure. The amplitude of ventricular contraction depends primarily upon the so-called initial tension and in the case of the right ventricle this tension is the resistance in the pulmonary circuit. After prolonged increased tension, the ventricular wall will become stretched and a point is reached where a complete contraction is impossible, thus giving way to an increased venous pressure.

Running along concomitantly with the aforesaid mechanism, the following events usually take place. First, hypertrophy and subsequent dilatation of the right ventricle occurs. Second, dilatation of the pulmonary artery with the development of a pulmonary conus visible by x-ray occurs with the eventual development of a pulmonary insufficiency and the development of a Graham-Steell murmur. Occasionally, the dilatation becomes so prominent as to produce dancing shadows at the hilum of the lung. Thirdly, concomitant with the sclerosis of the pulmonary artery there is sclerosis of the chambers of the heart which are subject to the increased tension. This

condition affects the left auricle only when the obstructive process is distal to the pulmonary circuit. As a rule the pulmonary veins are also involved by phlebosclerosis except in the case of emphysema. The left ventricle is not affected unless, of course, there is disease of the coronaries or of the greater circulation.

Eventually, when dilatation of the right ventricle becomes extreme, dilatation of the tricuspid ring increases with a subsequent tricuspid insufficiency. This insufficiency is enhanced by sclerosis of the valves and endocardium. This insufficiency causes a reflux of venous blood that brings about congestion in almost all of the viscera in the abdominal cavity. The visceral lesions are probably due to increased venous pressure rather than merely to congestion.

With further strain on the right ventricle dilatation progresses and more and more blood remains in that ventricle at the end of systole, thus causing the right auricle to empty against an ever increasing positive pressure and at this point the increase in venous pressure attains heights that produce obvious effects on the body. As long as the right heart is compensating no outstanding signs and symptoms are present but when a state of chronic failure ensues a host of physiologic and morphologic changes occur.

EFFECTS PRODUCED BY RIGHT SIDED HEART FAILURE

Arteriocapillary fibrosis progressively increases, the alveolar wall becomes greatly thickened and the alveoli decrease in size resulting in a decreased vital capacity. Reversion to the embryonal type of lung takes place. The blood flow is slowed, engorgement occurs, and the already present arteriosclerosis predisposes to infarct formation and consolidation. The result is an ever increasing anoxemia, cyanosis, and polycythemia. The exertional dyspnea seen in the compensated state is now orthopneic and hemoptysis is common. The pleura is affected in that hydrothorax is a common occurrence; however, its mechanism of production is a disputed question at present.

The outflow of blood through the hepatic vein is hindered by the increased pressure in the inferior vena cava. Increased pressure within the portal system is the result and congestion and enlargement of the liver ensues with eventual cardiac cirrhosis and the production of ascites. Some writers believe the cirrhosis to be due to replacement fibrosis induced by pressure atrophy on the central vein of the lobule while Moschcowitz and others believe it is the end result of phlebosclerosis caused by the long standing increased venous pressure—essentially the same process that occurs in the lung, but on the venous side in the liver. Ninty per cent of the cases show impairment of liver function; urobilinogen is usually increased in both the stool and in the urine; hyperbilirubinemia usually parallels the intensity of the right sided failure; and jaundice is seen to occur in the later stages.

At times the venules of the upper thorax become prominent owing to the collateral circulation between the pulmonary and bronchial veins. When pulmonary congestion is severe some of the blood from the right ventricle fails to go through the pulmonary capillaries but instead is shunted into the bronchial veins thence to the azygos and then into the internal mammary veins—the pressure thus being transmitted to the veins of the pectoral wall.

In the early stages of pulmonary hypertension the spleen in congested and markedly cyanotic. The venous sinuses are dilated and the organ is enlarged but not palpable. When failure ensues the spleen enlarges considerably but due to the concomitant fibrosis and contraction the spleen usually become smaller than normal. This explains why palpable spleens are rare in congestive failure. When the spleen is palpable in congestive failure, cardiac cirrhosis is usually prominent. Similar factors are present here as in the liver when cardiac cirrhosis is present.

The kidneys, associated with uncomplicated pulmonary hypertension, are very little, if any, affected. There may be seen a slight amount of intertubular increase in connective tissue. Obviously, if there is left

sided failure, the kidneys will exhibit much pathology. When right sided failure sets in, oliguria with a high specific gravity is first to be seen. The cause of oliguria is obscure but is associated with the shunting of fluid away from the kidney by the development of edema. Proteinuria of a considerable degree is observed and is due to increased permeability of the glomerular capillaries. Casts are usually present and azotemia is common and always associated with oliguria. Excretion tests show a decreased ability of the kidney to excrete dye and this, likewise, is due to the oliguria. As a rule, if the failure is overcome, the kidney will return to normal with very little lasting effect to be seen.

Little is known of the central nervous system in uncomplicated right sided failure. There are no marked anatomic changes noted. Edema is usually absent. There is obvious venous congestion and any rise in pressure in this system must be due to this engorgement. Cerebral disorders observed in right sided failure must be on a functional basis. Fishberg believes that the psychosis so frequently seen is due to dehydration.

Peripheral edema is one of the most prominent signs of right sided failure. At first the edema is latent and indirectly proportional to the amount of diuresis. It is believed that the edema, in the largest part, is due to the increased hydrostatic pressure in the venous end of the capillaries which overcomes the neutralizing effect of the colloidal osmotic pressure of the plasma. When the edema is of long standing, a hypoproteinemia results due to the loss of protein that has escaped into the subcutaneous tissues, contributing to the already existing peripheral edema. Proteinuria may further reduce the blood proteins, and also, an inadequate intake of proteins, or frequent tapping of exudates may lower the blood protein. Increased permeability of the capillaries due to anoxemia may be another factor; however, this is not believed by some investigators because the protein content of the edema fluid is low. It has been shown experimentally that dilatation of the capillaries is the rule in right sided failure and to a certain extent in compensated pulmonary hypertension.

Left sided failure following right sided failure is the exception rather than the rule. However, there are two factors which can explain a secondary left sided failure: (1) a sustained and progressive rise of effective venous pressure must eventually influence the work of the left ventricle. Stroke volume (Starling) is increased and leads inevitably to hypertrophy and dilatation. Now an increased left ventricular discharge is of necessity followed by increased discharge of the right ventricle so that a vicious cycle is set up that takes its toll upon both sides of the heart; (2) the anoxemia must increase the work of the left ventricle which is already overworked by an increased venous return. Such a heart requires more oxygen than normal but the amount is insufficient due to reduction in coronary flow that accompanies hypertrophy and dilatation of the left ventricular muscle. Another vicious cycle is set up.

CLINICAL PICTURE OF PULMONARY HYPERTENSION

There is usually a history of progressive dyspnea out of all proportion to other evidences of congestive heart failure. The dyspnea has as its basis ventilation which immediately suggests its pulmonary origin. Anoxemia occurs with a resulting cyanosis (blue hypertensive) and the patient tends to look older than his age. As the right heart decompensates, a tricuspid insufficiency arises and a markedly elevated venous pressure with an enlarged tender, and possibly pulsating liver is seen. Peripheral edema and ascites become outstanding features. Ascultation reveals an accentuated pulmonic second sound. A Graham-Steell murmur is heard when mitral stenosis is present along with marked "back-pressure effects." If the obstruction is proximal to the alveolar system, the lung fields will be clear. A systolic pulsation may be felt to the left of the sternum and is due to the hypertrophied right ventricle.

The laboratory affords a great deal of confirmatory evidence. X-ray reveals a prominent pulmonary conus, an enlarged

right ventricle and auricle, and usually clear pulmonary fields. "Dancing" hilar shadows may be observed by fluoroscopy. If the obstruction is at the mitral valve, the left auricle will be enlarged and the lung fields will be congested.

The venous pressure is increased and the circulation time is increased to a degree that approximately parallels the amount of decompensation.

Finally, the characteristic electrocardiographic pattern indicative of right ventricular hypertrophy and strain lends corroborative evidence. This consists of right axis deviation and inversion of the T wave in leads II, III, and IVF. There may be secondary polycythemia. The criteria for the diagnosis of pulmonary hypertension as set down by the New York Heart Association are:

(1) Radiologic evidence of undue prominence of the pulmonary artery and right ventricle; (2) right axis deviation of the QRS complex in the electrocardiogram; (3) marked dyspnea and cyanosis, and (4) polycythemia.

SUMMARY

Pulmonary hypertension has become a definite entity. This paper has reviewed the more important circumstances under which hypertension of the lesser circuit occurs. The compensatory mechanism and the resulting pathology to such a hypertension has been discussed along with the clinical syndrome observed in most cases. Even though pulmonary tension cannot be measured accurately, let us hope that in the future such an hypertension will be considered and, if possible, combated whenever there occurs a disease in which pulmonary hypertension is a distinct possibility.

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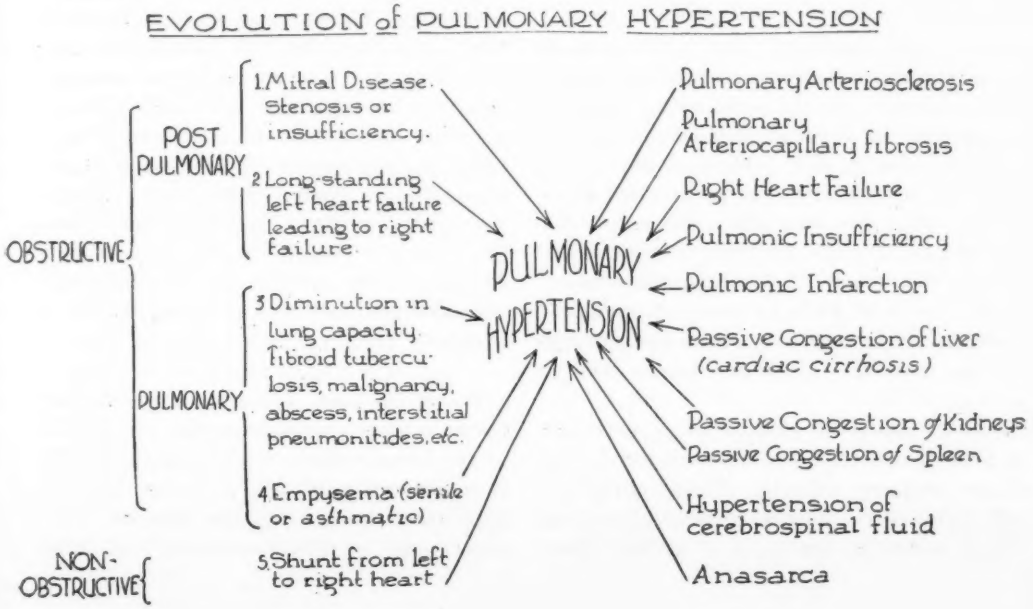

THE RELATION OF HYPERTENSION TO VASCULAR DISEASE

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INTRODUCTION

Probably one of the most fertile fields for investigation in the past ten years has been the study of hypertension. Interest in this field received enormous impetus by the reassociation of the kidney with hypertension, many years after the kidney was first incriminated by Bright, and relegated to a minor role by subsequent workers. This recent work has been productive of many new and interesting discoveries.

The more one reads the literature of hypertension the more he becomes lost in the conflicting maze of theories regarding the changes it produces in the structure and function of tissues. One finds many conflicting reports. For instance, some observers state that arteriosclerosis is one of the causes of high blood pressure; but it must be determined first whether arteriosclerosis is the cause of high blood pressure or hypertension is the cause of arterial degeneration. Moritz and Oldt[67] think that arteriosclerosis is a cause, while Castleman and Smithwick,[18] as well as many others, believe that in an uncomplicated degeneration of the arteries blood pressure does not rise.

In recent years, attempts have been made to subdivide essential hypertension into a large number of different types. Such a separation, in so far as it has been successful, hastened to cast further doubt on the validity of Bright's monogenetic hypothesis. Any one who bases his conclusion on the available clinical evidence alone must necessarily adopt the polygenetic theory. Such a point of view is, however, much too narrow. It is necessary that one surveys all the evidence—experimental as well as clinical.[68]

In this paper, the relation of hypertension to vascular disease will be considered from three primary aspects. First, pathologic variations of the normal physiologic actions which serve to maintain a normal blood pressure. Secondly, pathologic changes in various tissues and organs which are acknowledged to be secondary to hypertension. Last shall be considered the very pertinent, but extremely controversial, problem of the relation of renal arteriolar sclerosis to hypertension.

HISTORY

Hypertension was formerly regarded as always being an expression of either Bright's disease (nephritis) or arteriosclerosis. In 1874, Mahomed described the "prealbuminuric stage of Bright's disease", a term indicating that hypertension may precede frank nephritis but not disassociating the two. Von Basch, 1874, recognized that hypertension may exist in the absence of nephritis and demonstrable arteriosclerosis, and attributed it when present under these circumstances to "latent arteriosclerosis". Allbutt, 1896, differentiated hypertension ("hyperpiesia") from Bright's disease and senile arteriosclerosis. Huchard, 1899, also recognized hypertension in the absence of Bright's disease and, to emphasis its importance in the etiology of arteriosclerosis, called it "presclerosis." "Hypertensive cardiovascular disease" was the designation used by Theodore Janeway in 1913, and it became widely accepted at the time. Frank in 1911 introduced the term "essential hypertension." Volhard and Fahr (1914) and Keith, Wagner and Kernohan (1928), have especially directed attention to cases of essential hypertension which are characterized by kidney involvement and a rapidly progressive, fatal course—"malignant nephrosclerosis" or "malignant hypertension", in contrast to the commoner, "benign" disorder. The importance of cerebral vascular spasm in the production of symptoms was emphasized by Pal (1905) and discussed in detail by Oppenheimer and Fishberg (1928).

DEFINITION

Blood pressure is defined by Wright[117] as the lateral pressure exerted on the walls of the blood vessels by the contained blood. It consists of a systolic phase, which is the maximum pressure in the arteries during contraction of the ventricles, and a dia-

stolic phase, which is a measure of peripheral resistance depending mainly on the tone of the arteries. Hypertension or high blood pressure, according to Wiggers,[112] is a state in which arterial pressure exceeds normal values, taking into account sex, age, race, activity, and temporary psychic states. Generally no doubt as to the presence of hypertension can exist when pressure relations exceed the values 150/100 mg. mercury.

FACTORS WHICH COMBINE TO MAINTAIN THE NORMAL ARTERIAL PRESSURE

Normally there are five physiologic factors operating in the human body as blood pressure determinants: (1) The pumping action of the heart; (2) the peripheral resistance; (3) the quantity of blood in the arterial system; (4) the viscosity of the blood; (5) the elasticity of the arterial walls.[12]

(1). *The Pumping Action of the Heart*—The means by which the cardiac contraction exerts its effect upon the arterial blood pressure is, obviously, through the quantity of blood which it is capable of discharging into the aorta in a unit period of time, that is, upon the output of the heart per minute. When more blood is forced into the already filled arterial system, it cannot escape at once from the system in the same amount as it is thrown into the aorta, so the arterial walls become stretched. The pressure rises until the velocity of flow through the arterioles is great enough to balance again the outflow from the system with the inflow.

(2). *The Peripheral Resistance*—This resides in the arterioles and to a less extent in the capillaries. By far the greater part of the peripheral resistance of the circulatory system is constituted by the arterioles of the abdominal viscera—the so-called splanchnic area. Stimulation of the great splanchnic nerve which innervates the rings of muscle fibres in the walls of these vessels causes their constriction, and consequently a reduction in the outflow from the arterial system. The pressure will continue to rise until inflow and outflow are again balanced. In the absence of compensatory changes in the other factors concerned in the maintenance of the pressure this remains at the higher level so long as the constriction persists. Dilatation of the vessels, that is, reduction in peripheral resistance, will of course be followed by the opposite effect. When the vessels of the splanchnic area are fully dilated they are capable of accommodating almost all the blood in the body; in such an event the blood pressure would fall to zero.

(3). *The Quantity of Blood in the Arterial System*—In any closed system of rigid tubes fluid must fill it to capacity in order that a pressure can be developed within it. The arterial walls are distensible and elastic, and a certain degree of stretching of these must occur before any considerable pressure is created. The arterial system must be actually over-filled and the greater the extent of the over-filling the greater will be the blood pressure. Loss of blood, either of all its elements, as in hemorrhage, or of the fluid portion alone, if not compensated for sufficiently by readjustment of the other factors concerned in blood pressure maintenance, must inevitably result in a fall of pressure. Increasing the total amount of circulating fluid artificially as by the transfusion of blood or of a solution such as saline or gum will elevate the pressure again. Saline solution, however, causes a very evanescent rise, since it soon leaks from the vessels into the surrounding tissues. In animals the blood pressure may be lowered by hemorrhage to a third of its normal value and restored again to its original level by reintroducing into the circulation the blood which has been removed.

(4). *The Viscosity of the Blood*—The greater the viscosity or "thickness" of any liquid the greater is the pressure required to force it along a length of narrow tube in a given time; or if the pressure be constant the longer is the time required for it to traverse the tube. The frictional resistance which is developed between the parts of the liquid itself, that is, the internal friction is greater when the viscosity is high than when it is low. Viscosity depends upon the

degree to which the molecules or particles of a fluid cohere to one another. Blood is some five times more viscous than water. With regard to the influence of viscosity upon the blood pressure, it is again a matter of outflow through the arterioles. If the driving force remains constant and the caliber of the vessels is unchanged, then the greater the viscosity the greater will be the frictional resistance developed in this region and the less will be the quantity of fluid that will pass through in a unit of time.

(5). *The Elasticity of the Vessel Walls*—This is concerned mainly with the origin and maintenance of the diastolic pressure and with sustaining the mean pressure at a higher level than would be possible in a rigid system under otherwise identical conditions. The elasticity of arterial tissue does not come into play to any notable extent with a pressure below from 30 to 40 mm. of mercury. Below this level there would be little stretching of the walls of the arteries which would then behave like a system of rigid tubes. At the usual diastolic pressure that exists, however, the walls are stretched and by virtue of their elasticity tend to recoil against the distending force. The flow of blood is pulsatile in the arteries. Beyond the arterioles, that is, in the capillaries and veins the flow is continuous. The conversion of the pulsatile flow to an even one depends upon the existence of a diastolic pressure.[12]

The foregoing facts apply directly to the arterial system. The elasticity of the vascular walls and the peripheral resistance are both essential for the development of the diastolic pressure. As the contents of the ventricle are thrown into the already overfilled system during systole the added pressure which is then exerted upon the vascular walls causes their further distension. After the completion of systole the elastic walls rebound and pressing upon the blood within their embrace force it onwards through the peripheral vessels. In other words, the arterial lumen returns to its previous diameter and the energy that had been stored up during the stretching of the elastic tissue is in this way gradually expended during diastole.[12]

According to Wright,[117] blood pressure varies as a product of the cardiac output and the peripheral resistance. Cardiac output depends upon venous return, the force of the heart, and the rate of the heart. Peripheral resistance, on the other hand, is governed by the viscosity of the blood, the volume of the circulating blood, the velocity of flow, and the size of the lumens of the vessels. The last-mentioned factor is probably the most important in peripheral resistance. The drop in pressure of the blood as it flows through the arterioles is greater than for any other part of the vascular system. Thus, it is thought that these vessels comprise the greatest factor of peripheral resistance. The caliber of the arteriolar lumens is under the control of the vasomotor center by means of the autonomic nervous system. In addition, these vessels are affected directly by metabolites, hydrogen ion concentration, hormones (addenaline and pituitrin), and temperature (body and environmental). The vasomotor center is influenced by impulses from the higher cerebral centers, by carbon dioxide and oxygen concentrations in the blood, by radiation from the respiratory center, but especially by the nerves from the carotid sinus and the aortic arch. These nerves exert a reflex inhibitory action upon the vasoconstrictor center and are probably the most important mechanism for regulation of normal blood pressure, for in addition to their effect on the vasomotor center, they produce effects on the cardiac center in the control of heart rate, and also influence adrenaline secretion, which in turn affects the blood vessels directly. Thus, Heymans[52] considers the regulation of blood pressure as essentially and fundamentally an automatic proprioceptive reflex mechanism.

Besides originating from the carotid sinus and the cardioaortic area, pressosensitive reflexes also arise from the vascular territory of the celiac and mesenteric arteries, and from the territory of the thoracic and pulmonary arteries,[52] as well as from the right auricle and the venae cavae

at their entrance into the heart (Bainbridge reflex).

It is to be remembered too that the cerebral vessels and pulmonary vessels are supplied by sympathetic non-medullated vasomotor fibers and that they respond to impulses arriving through these nerves. The coronary circulation is supplied by vagus and sympathetic nerves, the vagus here conveying constrictor fibers and the sympathetic conveying dilator fibers.

PATHOLOGIC VARIATIONS OF THE NORMAL PHYSIOLOGICAL ACTIONS WHICH SERVE TO MAINTAIN A NORMAL BLOOD PRESSURE

(1). *Hypertension Due To Alteration in the Pumping Action of the Heart*—Hypertension due to alteration of this physiologic mechanism is characterized by three clinical findings: (a) increase in systolic pressure; (b) low or normal diastolic pressure, and (c) an obvious increase in pulse pressure.[97]

The predominant cause of increased blood pressure here is the increase in cardiac output, being in turn due to an increase in the minute volume of the heart. The minute volume is determined by two variable factors, that is: (1) the cardiac rate and (2) the amount of venous return to the heart. An increase in cardiac rate will not result in an increase in the minute volume of the heart without a concomitant adequate ventricular filling[12] which presupposes a greater venous return.[97]

Thyrotoxicosis,[95] simple tachycardia due to emotion, or excitement, and arteriovenous aneurysms[32] all result in an increase in venous return to the heart. Thyrotoxicosis and tachycardia exert their effect by speeding up the circulatory rate in the body. An arteriovenous aneurysm increases the cardiac return by mechanical means. An increase in venous return causes an increase in the pressure of the great veins leading to the heart, and, through a reflex mechanism (Bainbridge reflex), the heart is accelerated in rate. Thus, both factors upon which the minute volume of the heart is dependent are increased. Since there is no increase in peripheral resistances, the diastolic pressure remains normal; or, as in the case of thyrotoxicosis, becomes lowered due to peripheral vasodilatation.

Aortic regurgitation and heart block may cause an increase in systolic pressure through a somewhat different mechanism. In these two conditions there is no increase in pulse rate. The increase in minute volume observed is due only to the increased ventricular filling. In aortic regurgitation the incompetent aortic valves allow the reflux of blood from the aorta back into the left ventricle during diastole; the heart muscle fibers are stretched, react more forcibly (Starling's Law of the Heart), and systolic ejection is more complete.[12] Thus an increased diastolic ventricular volume due to a two-way filling (mitral and aortic) plus an increase in efficiency of cardiac muscle results in an increased minute volume and an increase in systolic pressure. The diastolic pressure in this condition is obviously lowered.[97]

Systolic hypertension associated with heart block has a very similar pathogenesis, the chief difference lies in the fact that the increased diastolic ventricular volume is due to the missing of a systolic period rather than to valvular incompetency.[97]

(2). *Hypertension Due to an Increase in the Viscosity of the Blood*—Rarely does this factor play a prominent role in the production of hypertension. Probably the only clinical condition in which the hypertension could be traced to an increased viscosity of the blood is polycythemia vera. Many claim that in the great majority of cases exhibiting hypertension and polycythemia, the hypertension is primary and that the increased red cell count is due to a mild cardiac decompensation. Tinney, Hall and Griffin,[103] however, have fairly well demonstrated that true polycythemia vera may increase the blood pressure. They found that 40 per cent of 163 proved cases with no demonstrable decompensation had an associated hypertension.[97]

With an increase in number of red blood cells there is an increase in viscosity of the blood and an increase in frictional resistance to flow. In the presence of an increased resistance, a constant driving force, and a constant size of the arterioles, there must result a decrease in outflow through

the arterioles. Hemodynamical laws demand that, under such conditions, the pressure within the arterial tree proximal to the arterioles must rise in order to force enough blood through the arterioles to equalize the arteriolar inflow and outflow.[12, 97]

This is the same mechanism whereby hypertension is produced when peripheral resistance is increased. The only difference lies in the fact that in the latter, arteriolar outflow is diminished by arteriolar constriction rather than by an increased frictional resistance. Similarly, also, the diastolic pressure is affected to a greater extent than is the systolic.[97]

(3). *Hypertension Due to a Decrease in Elasticity of the Arterial Walls*—Under this heading there will be considered only the larger, more elastic, arteries, such as the aorta and its immediate branches. Arteriolosclerosis, especially of the kidneys, and hypertension will be considered in another section of this paper.

It is a well known fact that, during the normal process of senescence, these vessels undergo atheromatous and calciferous changes; suffer a loss of elastic tissue; and thus lose a large part of their resiliency. As this process progresses, the large arteries concomitantly become less able to "give" with each systolic ejection of blood from the left ventricle. The systolic blood pressure, therefore, rises. In a similar manner these inelastic vessels lose the power of "clamping down" on the smaller diastolic volume of blood. The diastolic pressure falls, remains constant, or rises only slightly. Patients with systolic-diastolic ratios in the nature of 200/90 with little evidence of renal damage or cardiac enlargement, and with peripheral and fundal evidence of extreme arteriosclerosis, should exemplify this type of hypertension.[97]

Cerebral arteriosclerosis may produce an increase in blood pressure through the effect of cerebral ischemia on the vasomotor regulatory centers.

(4). *Hypertension Due to an Increase in Blood Volume*—Blood volume may be increased locally or generally. In either condition there will be an increase in both the systolic and diastolic pressures with a fairly normal pulse pressure.[97]

The localized increase in blood volume is exemplified in the condition of coarctation of the aorta.[14, 85, 86, 88] Here, the mechanical over-filling of the arteries of the upper extremities, head, and neck give rise to an increase in blood pressure in these areas with a decrease in pressure distal to the constriction.[97]

Steele and Cohn[98] report one case of coarctation with a generalized hypertension and suggest that, in some of these cases, the hypertension may be due to renal ischemia. They compare the hypertension in their case to that seen experimentally in constriction of the renal arteries.[43]

A generalized increase in circulatory volume is seen primarily with infusions[12] of hypertonic or protein-containing solutions, and clinically in pituitary and adrenal disorders. Some clinicians have obtained good results in the treatment of patients with essential hypertension by various methods aimed to decrease the blood volume.[97]

(5). *Hypertension Due to an Increase in Peripheral Resistance*—A great deal of experimental work has been done on the mode of production of an increased peripheral resistance, especially in renal diseases and the so-called "essential" hypertension. A survey of the literature in this respect leaves one confused in a maze of theories based upon only a few actual facts at the present time. Suffice it to say that early in all conditions associated with an increased peripheral resistance there is an arteriolar constriction which may be nervous or humoral in origin. In the presence of a constant driving force and an increased peripheral resistance, the pressure in the arterial system must arise in order to maintain the necessary relationship between arteriolar inflow and outflow.[97] Clinically this category is characterized by an increased systolic and diastolic pressure with a variable pulse pressure depending upon the relative degree of change of the two preceding factors.

Increased peripheral resistance associated with ischemia of vital organs, particularly the brain and kidney, may represent primary causes of hypertension. At this point, such conditions in the brain will be considered; those in the kidney will be dealt with later in the paper.

In any condition in which there is an increase in the intracranial pressure, such as in brain tumors[11] or cerebral contusion,[19] certain physiologic mechanisms come into play in order to preserve an adequate cerebral arterial supply. Practically speaking, the cranial cavity contains three fluids; that is, cerebrospinal fluid, venous blood and arterial blood. If for any reason the pressure within the cranium is increased; there is a compensatory decrease in venous blood and cerebrospinal fluid volume. The arterial supply to the brain does not decrease, however, for the pressure within the arterial tree is greater than that within the other two systems. There is, then, obviously an increase in cerebral congestion, since the arterial supply remains constant and the venous outflow decreases. A further increase in intracranial pressure thus takes place. When the intracranial pressure rises above the arterial pressure, a cerebral anemia results, with a concomitant embarrassment to the vital regulatory centers of the brain. In order for life to exist, the arterial pressure must rise. This rise is brought about through a stimulation of the medullary vasomotor centers with a resulting widespread vasoconstriction.[97]

PATHOLOGIC CHANGES IN VARIOUS TISSUES AND ORGANS, ESPECIALLY VASCULAR CHANGES, WHICH ARE ACKNOWLEDGED TO BE SECONDARY TO HYPERTENSION

(1). *Nervous System*—The vast majority of work indicative of possible functional changes in the nervous system has been experimental in nature. The indication seems to be that while there are disturbances in the normal physiologic function of the reflexes and nerves involved in blood pressure control, these disturbances are secondary to the hypertension and not the underlying cause. That a disturbance of the nervous mechanism exists is indicated by the fact that the natural relationship between pulse rate and blood pressure is reversed; rise of blood pressure as seen in hypertension being accompanied by increased pulse rate.[4] Ordinarily, of course, a rise in blood pressure sets up impulses from the carotid sinus and cardioaortic areas, which exert inhibiting influence of the cardiac and vasomotor centers, bringing about slowing of the heart rate and vasodilation, with consequent lowering of the blood pressure. In hypertensives, vasomotor release can be brought about by the application of heat and by novocaine injection of the nerves, so that there results increase in blood flow essentially the same as in normal individuals.[97] This indicates that there is essentially normal vasomotor activity and that the apparent changes are secondary in nature. Thus Pickering[81] states that "normal vasomotor activity is superimposed on an intrinsic vascular hypertonus which is independent of the vasomotor nerves." Further proof of the activity of the vasomotor nervous mechanism is found in the Hines-Brown test. In the light of what is said above, the response in this test peculiar to hypertensives or potential hypertensives may be considered as not a fundamental abnormality of the vasomotor nerves, but a response conditioned and determined by some other underlying factor.

Experimentally, hypertension has been produced by resection of the carotid sinus and aortic nerves.[52, 56] In dogs, the pressure may rise from 130 mm. mercury to 250 or 300 mm. mercury as a consequence of this procedure. This, of course, is due to the release of inhibition of vasoconstrictor and cardioaccelerator centers, the activity of which, under normal conditions, is permanently moderated reflexly by the aortic and carotid sinus nerves. This hypertension persists for a considerable length of time, but it is not permanent. It is prevented or caused to disappear by total excision of the paravertebral ganglionic chains from the stellate ganglia down to the pelvic ganglia.[52] This form of hypertension seems clearly quite different from permanent clinical hypertension, although it is thought by some that there is a type of "neurogenic

hypertension" that exists clinically and resembles this experimentally produced hypertension.

In this connection, it might be well to mention that many surgical procedures have been devised wherein various attacks are made upon different portions of the sympathetic nervous system. The effects of these operations on blood pressure are not constant, as reported by Allen and Adson.[5] It is claimed, however, that a relief of symptoms frequently results. That the blood pressure is inconstantly affected further indicates that the functional pathology is not primarily for the nervous system, but for some other factor, involving the nervous system only secondarily, if at all.

Special mention should be made of cerebral circulation and the reaction of pial arteries to increase in blood pressure. Fog[34] found that the usual response of these vessels (pial arterioles) to rises in arterial pressure is by constriction. Sometimes, no change in caliber occurs. This indicates that in these cases also an increase in tonus of the walls of the vessels has taken place. Without such active change in the walls, the sudden rise in pressure would cause passive dilatation. Thus, changes in the intravascular pressure cause changes in the physiologic state (tonus) of the walls of the pial arteries. This tonic regulation persists immediately after bilateral section of the cervical portion of the sympathetic trunk and bilateral section of the vasosensory nerves from the aorta and carotid sinus. The mechanism, then, by which the tonus of these vessels is regulated is not established —it is possibly by intracranial regulation or by local reaction of the smooth muscle of the arterial wall. This type of response to elevation of blood pressure further emphasizes that the cerebral circulation and the pial vessels are no exceptions to the general increase in intravascular pressure. The rise in pressure in the choroidal plexuses with the effect on the cerebrospinal fluid pressure becomes more clearly understood.

(2). *Cardiovascular System*—As stated in the discussion of normal blood pressure regulation, the factors mainly involved are cardiac output and peripheral resistance. Blood volume and the viscosity of the blood are also of some importance, but mainly as factors influencing peripheral resistance. Since hypertension is usually considered as a functional disease of the cardiovascular system, we look for most changes in function and associated structure in this system.

(a) The Heart: The typical heart of essential hypertension shows, pathologically, hypertrophy of the left ventricle with or without associated dilatation.[49, 55, 110] It may be said that a heart weighing over 500 grams is from a case of essential hypertension if other conditions leading to cardiac hypertrophy are absent (valvular disease, glomerulonephritis, hyperthyroidism, adherent pericardium, syphilitic aortitis). These changes, however, are secondary in nature. The heart could be involved primarily only if cardiac output were increased. It has been shown by some workers[101, 110] that the cardiac output is not increased in essential hypertension. In addition to studying cardiac output in hypertensive human beings, Holman and Page[54] investigated the subject in dogs, in which hypertension has been produced by renal artery constrictions. It was found that the bringing on of hypertension did not change cardiac output.

The increased size of the heart and the increased resistance against which it must work in hypertensives bring up the questions as to its efficiency and the functioning of the coronary circulation, since, in the benign type of essential hypertension, death is frequently by heart failure. Wright, Hallaran, and Wiggers[116] have made a study of the "effort index" for hearts of hypertensive subjects. In a comparison of the efficiency of the left ventricle in hypertensives and in normal individuals, they concluded that the left ventricle in hypertension maintains an "efficiency quotient" as good or even better than that of the normal left ventricle, by virtue of a large pulse pressure in spite of an elevated dias-

tolic pressure. Thus, according to Wiggers,[113] physiologically the action of the left ventricle is not impaired as a result of the greater work imposed upon it by severe hypertension. He states further that, in animals, the mechanical efficiency actually increases and the economy with which the mechanical energy is utilized to propel blood is decidedly increased. However, in order to do this, the left ventricle operates continually in the region of cardiac reserve and, if additional distention occurs, for example, because of an added return of blood, (as during exercise) it responds with decreased rather than, as normally, with increased output. This explains the limited capacity for exercise and the development of the "effort syndrome" on exertion.

As regards coronory circulation and blood supply to the heart, it is known[10,113] that coronary inflow increases as does the systolic pressure, and that mechanical adaptations between peripheral coronary resistance and aortic pressure, rather than nervous reflexes or chemical action, are chiefly concerned. It is pointed out by Tollman,[104] however, that, as noted above, the heart undergoes hypertrophy to meet the increased load in hypertension. This hypertrophy increases the mass and diameter of the individual fibers; there is no increase in the number of fibers. The increase in size and diameter of the fibers introduces a metabolic disadvantage, impairing the state of cardiac nutrition. In addition, the frequently concomitant coronary sclerosis may prevent the necessary increase or even maintenance of cardiac circulation. The result is heart failure and a cardiac death.

(b) The Blood: The physical and chemical properties of the blood are important as possibly being involved in the abnormal function of hypertension. Volume and viscosity are factors which, if increased, could be responsible for elevation of the blood pressure. Linder, Lundsgaard, Van Slyke, and Stillman[63] and Austrian[7] have shown that in arterial hypertension both blood volume and blood viscosity are within normal range. In studying dogs with hypertension produced by the Goldblatt technic, Gibson and Robinson[40] found irregular changes in the blood volume. They concluded that there was apparently little relationship between blood volume and hypertension in these dogs.

The most important possible change in the chemical makeup of the blood in hypertension is the presence of substances which may exert a vasoconstrictive influence, the "renal pressor substances"[77]; however, it is beyond the scope of this paper to consider this subject.

(c) The Blood Vessels: From the above data, it is seen that three of the factors (cardiac output, blood volume, and blood viscosity) which might influence blood pressure physiologically, are found to be largely normal in essential hypertension. This leaves only peripheral resistance as the one involved. It seems clear then that there is a fundamental increase in peripheral resistance in hypertension. In fact, Wiggers[113] states that calculations of peripheral resistance by several formulae show that it is increased in various forms of human hypertension. But the magnitude of the increase does not necessarily correspond to the degree to which mean blood pressure is elevated,[39,60] suggesting that other mechanisms are also concerned.

Peripheral resistance, as mentioned in the discussion of normal blood pressure, is largely determined by the size of the lumina of the arterioles. Pathologically, the essential and universal arterial change in primary hypertension is a muscular hypertrophy, which is proportional in degree to that of the left ventricle.[49] It involves the media chiefly, but the intima also shows reduplication of the internal elastic lamina, and a notable development of new musculoelastic layers. Kernohan, Anderson and Keith[61] excised small portions of muscle and determined by study of sections that there was an increased thickness of the arterial wall in proportion to the diameter of the lumen of the vessels in hypertension. These changes are almost surely secondary, and represent a physiological response to the increased intravascular tension.

According to Wiggers,[113] organic lesions are not generally widely enough distributed to account for the increased effective resistance. The only plausible view, then, is that the increased peripheral resistance is due to narrowing of the lumina of the vessels by functional contraction or vasospasm. The arterioles and prearterioles are probably most involved, though Wiggers feels that we should not be too dogmatic in assigning the increased resistance entirely to these vessels—some of the resistance may occur in vessels larger than prearterioles.

The work of Prinzmetal and Wilson[82] indicates that peripheral resistance is elevated by means of widespread hypertonus of the arterioles. They studied the blood flow through the limbs of patients with hypertension and found normal values. If only the splanchnic area were responsible for the increased resistance, vasoconstriction here and not in the periphery (muscles) would lead to a greater blood flow through the periphery. These findings are in keeping with the histologic changes in the arterioles of voluntary muscle. Furthermore, blood vessels of hypertensives are capable of dilatation, indicating that the increased peripheral resistance in hypertension is due to generalized vascular hypertonus and not to primary changes in the blood vessels.[97] It has been shown that, with slow injection of dilute epinephrine intravenously into hypertensive persons, the magnitude of the rise in blood pressure (systolic) is the same as in normal individuals.

The role of the larger arteries in the dynamics of hypertension is discussed by Wiggers.[113] He states that diminished elasticity of the aorta and its immediate branches accentuates the elevation of systolic pressure and reduces the diastolic pressure. It accounts also for the large pulse pressure and the rapid diastolic decline of the subclavian pulses in hypertension. An absence of a significant increase in diastolic pressure, far from denoting inconsequential involvement of the peripheral vessels, is probably more often a sign of an extension of vascular changes to the larger vessels. Although sclerosis of larger vessels produces these changes, the suspicion has existed that aortic distensibility is also decreased, even when no pathologic changes are demonstrable. The view that, in such subjects, the aortic distensibility is decreased beyond that common for corresponding ages is supported by considerations arising out of new experiments on circulation models, on the perfused dog's aorta in situ, and by simultaneous records of aortic pressures and diameters of living dogs. These views of Wiggers seem important as additional factors in the pathological physiology of hypertension.

(3) *The Retinal Fields*—The changes here observed ophthalmoscopically are mainly in the retinal arterioles. For that reason, this consideration is taken up immediately following the discussion of the blood vessels. The early vasoconstriction and subsequent structural changes in the arterioles can actually be seen here. The importance of parallelism between the retinal and cerebral vessels is pointed out by Gresser[47] and Salus.[87] According to Wagner[69] the earliest changes observed in the retinal vessels are those of increased arterial tonus, in which the lumen of the arterioles is narrowed but uniform. There is changed or altered relationship between the caliber of the arteriole and the vein as compared with the caliber of those structures in normal subjects. Following this, there is the beginning of recognizable organic change in the arterioles—there is hypertrophy of the tunica media, which causes narrowing of the lumen and gives it an irregular appearance. There is greater reflexion of light from the wall of the arteriole giving it a coppery or steely appearance. Since the veins and arterioles are in the same sheath, the increase in thickness of the arteriolar wall and the increased intra-arterial pressure produce indentation in the more easily compressible veins, the sign designated as arteriovenous compression. Finally, there is what probably represents the beginning of the first recognizable phase of true arteriosclerosis. There is more marked con-

traction and irregularity of the lumens of the arterioles, and exaggeration of the light reflex, associated with increased visibility of the vascular wall, and at times with thickening of the vascular sheaths. In a more advanced stage, in the smallest visible arterioles, there is an appearance of obliteration, often with fine punctate white areas of hyaline or fatty degeneration and punctate hemorrhages. There may be retinitis, characterized by cotton-wool patches or edema of the nerve fibers distal to the points of arteriolar narrowing, occlusion or spasm. Mild edema of the retina and flame-shaped hemorrhages are usually present. There is often edema of the optic disks.[105]

It is pointed out by Salus[87] that the process seems to start in the more peripheral divisions (capillaries and pre-capillaries) and pushes centralward while the disease progresses. Thus, the central artery and its main branches may be still wide while the smaller branches are constricted.

(4) *The Pulmonary System*—The functional disturbances on the pulmonary system in hypertension are entirely secondary. According to Wiggers,[112] a study of heart lung preparations indicates that pressure rises almost proportionately in the pulmonary artery and in the right ventricle and auricle. In man, the blood content of the lungs is increased. These facts are caused by: (a) increased venous return via the coronary system; and (b) increased collateral flow into the pulmonary capillaries via anastomosing bronchial vessels. Thus, vital capacity is reduced as a result of the pulmonary congestion.

Sympathetic non-medullated fibers end (in large numbers) in the media of the pulmonary arterioles and may be fairly safely presumed to be vasomotor in function (Wright[117]). Adrenaline injection and sympathetic stimulation produce vasoconstriction. The pulmonary blood pressure may be raised as much as 40 per cent by sympathetic stimulation, suggesting that the nervous control may be of considerable significance. In any event, whatever factors are responsible for the increased tonus of the splanchnic and peripheral vessels no doubt have an influence on the pulmonary and bronchial vessels with increase pressures here.

THE RELATION OF RENAL ARTERIOLOSCLEROSIS TO PRIMARY HYPERTENSION

Three views of the relationship between renal arteriolosclerosis and primary hypertension have been advocated: (1) that arteriolosclerosis causes hypertension; (2) that hypertension causes arteriolosclerosis; and (3) that the two processes originate independently and only intensify one another when associated together.[10]

The role of the kidneys in the production of hypertension has been under investigation since the time of Bright. That disturbance in kidney function might lead to elevation of the blood pressure has long been known. This formed the basis for the classification of hypertension into renal and non-renal types. Essential hypertension has for many years been considered as the non-renal hypertension, and thus is defined by Fishberg[32] as persistently elevated blood pressure which, on neither clinical nor anatomical grounds, can be considered due to inflammatory or obstructive renal disease. The work of Goldblatt, begun in 1923, has reopened the field and the kidney again is presented as the seat for the functional disturbances seen in hypertension.

It seems pertinent at this point to discuss briefly Goldblatt's famous experiments,[42,43,46] which have consisted in the use of silver clamps whereby the main renal arteries could be constricted and their lumina reduced to any desired caliber. This reduces blood flow through the kidneys. Ischemia of the kidneys is produced and with the ischemia, both systolic and diastolic blood pressures are elevated. When constriction of these arteries is moderate, there is no accompanying disturbance in renal function, detectable by the usual studies of urine and blood. In long standing hypertension of this type, intimal degenerative changes in the retinal arterioles and medial thickening of arterioles in other organs are observed. If constriction is made severe or complete from the begin-

ning, disturbance in renal function usually accompanies the elevated blood pressure and uremia may develop. In some of these experimental animals, there develop widespread fibrinoid and hyaline degeneration and necrosis of the arterioles, with petechiae in some organs, similar in all respects to the lesions observed in the acute malignant phase of essential hypertension in man. Such similarity then to the pictures seen in both benign and malignant essential hypertension in man make this work especially important as indicative of the functional changes in this condition. The view that the kidney may play a primary part in the development of essential hypertension is reenforced.

In order to attempt to clarify the controversial subject in question, it will probably be well to consider briefly the latest experimental studies of several of the outstanding workers interested in the relation of renal arteriolosclerosis to hypertension.

Bell[10] attacks the problem in this manner: If it is true that renal arteriolosclerosis causes hypertension, then all cases of hypertension should show arteriolosclerosis, and one might also expect that all cases of arteriolosclerosis would show hypertension. As a means of studying this problem, microscopic studies of the kidneys were made from 267 nonhypertensive individuals, all over 40 years of age. There were no hypertensive symptoms, the hearts were of normal size, and the systolic blood pressure was always below 140 mm. mercury. All cases of diabetes were excluded. In this control group 17.8 per cent showed renal arteriolosclerosis of grade 1 or higher, and 80 per cent were grade 1. When the cases were arranged with respect to age, it was found that arteriolosclerosis (grade 1 or higher) occurred in about 9 per cent of those between 40 and 60 years old, and in about 30 per cent of those over 70 years old. It is clear, therefore, that renal arteriosclerosis is an age change and that it occurs in a great many elderly individuals who do not have hypertension. If arteriolosclerosis is the only cause of hypertension, it is not clear why these individuals with arteriolosclerosis did not have an elevated blood pressure. It cannot be argued that hypertension was the cause of the arteriolosclerosis in these controls.

In autopsy studies, renal arteriolosclerosis has been found to be about four times as frequent in hypertensive persons as in nonhypertensive controls, but a great many typical cases, especially of the myocardial and coronary forms, do not show arteriolosclerosis. Those who insist that hypertension is always caused by renal vascular disease maintain that these cases without arteriolosclerosis have lesions in larger arteries sufficient to cause renal ischemia. However, sclerosis of large and small renal arteries is present in the majority of nonhypertensive elderly persons, and some of the hypertensive subjects have no disease even of these larger vessels.

Bell has studied the kidneys microscopically in about 600 persons with diabetes. It is well known that generalized arteriosclerosis is much more severe in elderly persons with diabetes than in persons without diabetes of corresponding age. Renal arteriolosclerosis is also very prominent in persons with diabetes and occurs in nearly half of those with small hearts and systolic pressures below 140 mm. mercury. Bell has found the incidence of arteriolosclerosis in diabetes to be greater with increasingly higher levels of systolic blood pressure.

It is obvious that there is a causal connection between hypertension and renal arteriolosclerosis in diabetes, but it is noteworthy that either condition may exist without the other. Age is a factor of great influence, since renal arteriolosclerosis was found in 31.8 per cent of persons with diabetes under 50 years of age and in 80 per cent of those over 50 years old.

The available data from Bell's studies indicate strongly that renal arteriolosclerosis is primarily an age change in the arteries and that when it develops to a sufficient degree, it usually brings about or intensifies hypertension, but in a great many instances it does not cause hypertension. Typical clinical hypertension may develop in the absence of renal arteriol-

osclerosis. It appears, therefore, that renal arteriolosclerosis and hypertension may originate independently, but when the vascular lesion becomes severe, it usually causes hypertension.

The evidence that hypertension causes renal arteriolosclerosis is not convincing. One argument against this view is the frequent absence of arteriolosclerosis in hypertensive disease of long duration. In chronic glomerulonephritis with hypertension for many years, true renal arteriolosclerosis is not often seen. The sclerotic arteries in this disease are usually examples of disuse atrophy. It is clear, however, that hypertension causes lesions in the retinal arteries, since these are not present at first and appear only after the blood pressure has reached high levels. It is easily possible that hypertension hastens the process of sclerosis elsewhere in the vascular system.

Moritz and Oldt[67] have recently made an extensive study of the arteriolar changes. They found that the objective examination of arterioles in all parts of the body of 100 control cases and 100 cases of chronic hypertension disclosed only one situation in which the presence of arteriolar sclerosis was almost invariably associated with hypertension and where the absence of arteriolar sclerosis almost invariably betokened an absence of high blood pressure. This was in the kidneys. Renal arteriolar sclerosis was present in 109 of the 200 cases studied, and 97 of these 109 proved to be cases of chronic hypertension. No comparable correlation could be found in any other organ or tissue.

They felt that these facts, together with the information gained from a study of the histologic characteristics of arteriolar disease in hypertensive and nonhypertensive individuals, supported the conclusion that renal arteriolar sclerosis is the most common cause of chronic hypertension. This conclusion is in accord with the recent demonstration by Goldblatt that chronic hypertension is regularly produced in dogs and monkeys by reducing the blood flow through the kidneys (renal ischemia). They believe that the effect of the renal arteriolar sclerosis in human hypertension appears to be the functional analogue of the renal arterial clamp in experimental hypertension. In both instances, hypertension does not necessarily lead to a sufficient degree of ischemia to impair renal function measurably.

They concluded that the only significant site of arteriolar sclerosis so far as the causation of hypertension is concerned, is the kidney.

Castleman and Smithwick[18] were afforded an unique opportunity to examine grossly the kidneys of hypertensive patients and to take biopsies therefrom, by the introduction of the surgical treatment of hypertension by dorso lumbar sympathectomy. From 1941-43, these workers collected renal biopsies from more than 100 hypertensive patients.

The 100 patients in this series ranged from 18 to 56 years of age, the average being 39. There were 43 males and 57 females. Their symptoms included headache, fatigue and occasionally visual disturbances. These had been present from one month to 15 years—average six years. The average systolic pressure was 210 and the average diastolic 130. Renal function determined by the PSP test showed that 60 per cent had normal function, that 20 per cent had a moderate reduction in excretion of the dye and that 20 per cent had a decided reduction.

Each biopsy was graded according to the severity of its vascular disease without knowledge of any clinical data about the patient, and it was found that there were enough differences in the degree of vascular disease to classify the group into five grades: grades 0, 1, 2, 3 and 4. The biopsies showed that a very few patients, usually the older ones, had an occasional focal scar with vascular disease in it. These scars are seen so often in nonhypertensive patients that it was felt that they should not influence the grading of the generalized vascular condition. This policy was also employed by Moritz and Oldt.

The most striking result of this work is the finding of such a high percentage of renal biopsies in which there is no or only minimal vascular disease. Seven per cent were graded 0, 21 per cent grade 1 and 25 per cent grade 2, that is, 28 per cent and possibly 53 per cent of the cases showed renal vascular systems so slightly damaged that it seemed unlikely that the blood flow could have been embarrassed sufficiently to be the one factor responsible for the hypertension. A valid objection might be raised that biopsy as small as the ones removed in this series is not an adequate sample of the whole kidney. It is believed, however, by Castleman and Smithwick that since great care was taken to select a representative region for biopsy and since similar morphologic findings were present in both kidneys in those 25 cases in which bilateral biopsies were taken, each biopsy is a fair sample of the renal tissue in the respective case.

When one considers that seven cases in this series, some with long standing hypertension, had no renal vascular disease at all, grade 0, one is led to believe that the hypertensive state antedates the vascular disease. It can further be stated that, in many of the 25 cases presenting grade two renal vascular disease in which the changes did not appear severe enough to have diminished the vascular bed to cause hypertension, the hypertension may also have antedated the vascular disease. This is in keeping with Cox and Docks' recent perfusion experiments on kidneys postmorten in which they found that "most kidneys from patients with hypertension without uremia have vascular beds in the normal range."

SUMMARY

In this paper, material under the following headings has been considered:

1. Factors which combine to maintain the normal arterial pressure.

2. Pathologic variations of the normal physiologic action which serve to maintain a normal blood pressure.

A. Hypertension due to alteration in the pumping action of the heart—thyrotoxicosis, tachycardia, aortic regurgitation, and heart block.

B. Hypertension due to an increase in the viscosity of the blood—polycythemia vera.

C. Hypertension due to a decrease in elasticity of the arterial walls—arteriosclerosis.

D. Hypertension due to an increase in blood volume—coarctation of the aorta.

E. Hypertension due to an increase in peripheral resistance — associated with ischemia of the brain.

3. Pathologic changes in various tissues and organs, especially vascular changes, which are acknowledged to be secondary to hypertension.

A. Nervous system.

B. Cardiovascular system.

(1) The heart.

(2) The blood.

(3) The blood vessels.

C. The retinal fields.

D. The pulmonary system.

4. The relation of renal arteriolosclerosis to primary hypertension.

A. Brief historical review.

B. Recent experimental work.

CONCLUSIONS

1. Certain pathologic variations of the factors controlling normal blood pressure can produce hypertension.

2. The nervous system, cardiovascular system, retinal fields and pulmonary system frequently show evidence of damage secondary to hypertension.

3. A state of hypertension not infrequently exists in man in which evidence of renal vascular disease is either absent or is insufficient to explain the elevated blood pressure. The implication is that in many cases some other functional factor or factors exist which are primarily responsible for the hypertensive state and which precede the appearance of renal vascular disease. It is certainly very probable that the vascular disease, once established, aggravates the already present hypertension, which in turn accelerates the arteriosclerosis—vicious circle. This is the stage in which the kidney is found postmortem and on which the theory of the primary

vascular origin of essential hypertension is based. Observations of the kidney biopsies in the earlier stages of hypertension have given the missing link to help substantiate the theory that the hypertensive state precedes the renal vascular disease in many cases.

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GASTRIC POLYPOSIS

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INTRODUCTION

Gastric polyposis is indeed a rare condition; however, its rarity does not preclude a knowledge of the subject. Gastric polyposis should constantly be considered in the differential diagnosis of gastric carcinoma, gastric ulcer, other benign tumors of the stomach, and possibly should be watched for in patients who have pernicious anemia. Therefore, it is the purpose of this paper to acquaint the reader with the theories of etiology, the histology, signs and symptoms, diagnosis, therapy, and prognosis of gastric polyposis so that he will be better able to cope with the vague gastric complaints with which he will be faced upon entering the practice of medicine.

DEFINITION

Galen originated the term polyp for describing pedunculated tumors of the nasal mucosa, it refers not to a structural property but merely to the form which may be assumed by any growth. Gastric polyposis then is the occurrence of multiple benign polypoid growths of the gastric mucous membrane involving diffusely the gastric mucosa.

HISTORY

The first gastric polyp is said to have been described by Amatus Lusitanus (1557). However, the diagnosis is not clear, and no autopsy was done. In 1761 Morgagni described a gastric polyp in a woman fifty years old who had died of dropsy. Cruveilheir (1835) wrote of the possibility of malignant change in polyps; he also wrote of the possibility of their obstructing the pylorus. Quain in 1857 reported a case of a nineteen year old girl who vomited a polyp with its pedicle. Rokitansky (1861) described gastric polypi in his textbook, and in 1864 Ebstein gave a detailed review of the literature of that time. In 1888 Menetrier gave his classical paper on gastric polyadenoma. In 1908 Wegele made the diagnosis of gastric polypi when he found a small polyp in the lumen of a stomach tube he had used in examining the patient. The diagnosis was made in a similar manner by Meyer and Chosrojeff in 1909. In 1911 Heinz made the diagnosis when he found a piece of adenomatous tissue in stomach washings; this was confirmed at operation. In 1912 the first roentgen film of gastric polyps was taken. In 1913 a case of polyadenoma associated with carcinoma was reported. During this same year Meulengracht used the term gastritis polyposa and brought careful histologic evidence to support the view that polyps were the result of chronic gastritis. In 1921 Eusterman and Senty presented a paper on the radiologic findings. The introduction of the gastroscope, at this time, as an aid to roentgen diagnosis, was a tremendous step forward and its use becomes constantly more important.

INCIDENCE

The relative frequency of this condition is difficult to estimate, since there are no complete statistics available, and since much of the early data on this subject are not reliable. Also because of the lack of symptoms many cases are overlooked. There were only 84 cases reported in the literature from 1820-1926, and only 41 cases reported from 1926-1941. This is probably not due to an actual increase of the condition itself,

but rather to an increase in the diagnostic acumen of the modern physician, and his willingness to report.

Two-thirds of the cases of gastric polyposis are discovered in the fourth and fifth decades; however, cases have been reported in individuals as young as 23 and as old as 80. The condition is more frequent in men than women, the ratio being approximately three to two.

ETIOLOGY

There are two recognized theories regarding the etiology of gastric polyposis, one is the congenital, the other the inflammatory. In the former it is believed that the tumors are of congenital origin, being true neoplastic growths arising from cells in the gastric mucosa which have retained their embryonic properties which are usually lost, especially that of proliferation. The upholders of this theory ascribe the inflammatory reaction usually found around such tumors to mechanical injury. In the latter theory it is believed that the growths arise from a chronic inflammatory gastritis, and Konjetzny claims to have found all the stages from hypertrophic gastritis to adenoma and finally to carcinoma.

Evidence for the congenital origin of gastric polyposis may be summed up as follows: The epithelial structure of the polypi is often irregular and atypical suggesting new growth, and the undifferentiated cuboidal cells are suggestive of an embryonic structure. The occurrence of the disorder in twins, three, four, and six siblings, and in a mother and son seems to support this contention. Occasionally a case has been reported in the newborn, also the hereditary factor is observed in diffuse alimentary polyposis of children, which may involve the stomach, duodenum, and colon. Weschselmann noted outspoken familial predisposition in 50 to 60 per cent of the cases in which the whole gastrointestinal tract was involved; however, Brunn and Pearl in a series of 135 cases found not one case in which there was an unquestionable positive hereditary factor. Another point in favor of this theory is the fact that the gastric polypi are indistinguishable anatomically from the intestinal variety. Still other points are the facts that evidence of inflammation is often wanting, that there is a clear margin between normal and abnormal tissue, that gastritis is common and polyps are rare.

Along experimental lines, proof of the congenital theory is given by Stewart who has reported the occurrence of diffuse lesions, characterized by adenomatous, hypertrophic, and hyperplastic overgrowth of the glandular rugae, in the pyloric chamber of almost everyone of his strain I mice. This is definitely inherited as a recessive character.

In favor of the inflammatory theory are the following: Gastritis though usually followed by atrophy, may cause hyperplasia of both epithelial and connective tissue; in certain areas of polyposis evidences of a preceding gastritis may be found; however, this evidence is usually wanting. Though polypi may be found in infancy, those of the most clinical importance usually seem to evolve in later life.

Experimentally, in animals, polypi have been produced by chronic irritation. Japanese workers have produced polypoid lesions by the injection of coal tar into the gastric mucosa of rats. Tsukiaskas found marked epithelial hypertrophy in the stomachs of two monkeys, probably produced by the irritation from ascaris. Fibiger fed a certain nematode to rats, and produced in the stomach mucosa wide-spread polypoid growths, the initial stages of which were epithelial hyperplasia and inflammation; he was able to follow the lesion to malignant degeneration and unquestionable metastasis. Fibiger believed that these gastric changes were produced by toxins liberated by the nematode. Wassink confirmed this work. Otto, reports a case, in man, in which a thick piece of wood was swallowed, causing marked gastritic symptoms for a year, and finally resulting in death. At necropsy a large gastric adenoma was found, which on section showed wood in its center. Zabel found large numbers of

megastoma intestinale in the tufts of a large gastric papilloma.

Some doubt is cast upon the theory of inflammation by the statistics of Lange who found no polypi in 46 cases of chronic gastritis but only slight hyperplastic phenomena. Also Perinin in 50 cases of chronic gastritis, in tuberculous subjects, found no evidence of polypi.

There are other lesser theories of origin of gastric polyposis. Some authorities believe the cause to be a primary degeneration of the glandular epithelium, not congenital, and the inflammatory changes to be secondary; and that the polypoid form is the result of mechanical pull by the action of the stomach on a growth already present. There are also those who believe the growths to be due to such causes as chronic alcoholism, arteriosclerosis of the gastric vessels, syphilis, amyloid degeneration, and dietetic errors, but these theories are not generally supported.

Brunn and Pearl are of the opinion that there are two types of epithelial overgrowth, an hyperplasia secondary to inflammation, and an hypertrophy congenital in origin. They found on studying sections taken from a large number of cases which conformed more to the congenital type, that the gastric epithelium assumed the dominant role, the connective tissue being relatively passive, and that evidence of chronic inflammatory changes is distinctly secondary.

Some authorities have combined the two main theories of origin concluding that certain cells are congenitally disposed to overgrowth and that inflammatory stimuli may be precipitating factors. This suggestion does something to account for the discrepancy between the frequency of gastritis and the rarity of polyps.

At the present time a great deal of interest is being paid to the relationship between gastritis, adenoma, and carcinoma, as first reported by Konjetzny. The high incidence of achlorhydria in cases of gastric polyposis indicates that the condition is associated with widespread changes in the gastric epithelium. In over 60 per cent of the cases of gastric carcinoma similar types of secretion have been reported. Schlindler in the gastroscopic examination of 2167 patients discovered 36 cases of multiple and single adenoma, 26 of which had a definite gastritis; of this number 20 were atrophic, two superficial, and four hypertrophic in type. Conversely 0.83 per cent of 1,814 patients without atrophic gastritis had visible adenomas, whereas 6 per cent of 353 patients with atrophic gastritis had visible adenomas, whereas 6 per cent of 353 patients with atrophic gastritis had adenomas.

The high incidence of gastric achlorhydria found in cases of gastric polyposis has brought about the question of the relationship between this condition and pernicious anemia. Schindler found gastric adenoma in six of 43 patients suffering from pernicious anemia, this being a much greater incidence than that found in patients not having pernicious anemia. Similarly, Rhoades found that gastric polyposis was more commonly found in persons dead of pernicious anemia, than those dead of other causes. In a postmortem study of patients dead of pernicious anemia Brown found chronic gastritis commonly present. She noted in 151 autopsies on such patients that the incidence of benign gastric tumors was 8 per cent, whereas the incidence in 18,200 patients without pernicious anemia was 0.03 per cent.

PATHOLOGY

The first classification of gastric polyposis was made by Menetrier in 1888. He described two types—polyadenome polypeaux, and polyadenome en nappe. In the former there are many discrete polyps, varying from one to several hundred in number, and scattered over the gastric mucosa. They are usually pedunculated, and may bear cysts. In the latter type the hypertrophy forms a well demarcated plaque, not cystic or pedunculated. It is composed of closely placed folds of hypertrophic mucous membrane resembling, in many instances, the convolutions of the brain. Menetrier believed the polypeaux

variety to be due to hypertrophic changes involving the ducts and necks of the glands, with the stalk and tumor pattern formed by the connective tissue elements derived from the submucosa; the en nappe type is thought to be more purely epithelial growth which involves the fundus of the glands.

Pearl and Brunn are of the opinion that the polyadenome polypeaux conforms to the type which is considered as congenital, whereas the polyadenoma en nappe corresponds to the description of a localized hypertropic gastritis. Menetrier, however, was of the opinion that both types were due to chronic irritation.

Thus, it is generally recognized that there are two types of gastric polyps—the congenital, neoplastic adenoma, and the acquired hyperplasias due to chronic irritation. It is frequently impossible to differentiate these two types of polyps on gross examination. Grossly the congenital type are soft polyps of various sizes. They are usually pedunculated but may be sessile, and they usually have a tufted or tree-like appearance. They are freely movable on the submucosa and each tumor is separated by an area of normal mucosa. Inflammatory processes are slight or absent. The mucous membrane away from the polyps may be normal or show an atrophic gastritis with reduction in gland tissue and achlorhydria. On section the connective tissue, which originates from the submucosa, seems to invaginate itself into the growth as a central fibrous tissue core. This connective tissue pushes the muscularis mucosa before it and the latter splits into strands as the connective tissue enters the base of the polyp. The connective tissue may be edematous containing thick or thin collagenous bundles. Resting upon the central core is the glandular epithelium which is arranged in a disorderly fashion into glands which show a considerable variation from the normal. Many are elongated, tortuous and dilated; some have been changed into small cysts containing old secretory material. At times the epithelial lining shows proliferation in the form of atypical papillae. Superficial ulceration, necrosis, and inflammatory infiltration often occur. The epithelial cells may be of the undifferentiated, tall hyperchromatic type, or of the type resembling the cells of the small intestine. There may be a mixture of goblet cells, Paneth cells, and the fuchsinophil cells of Lubarsch, with variations from one to another, even in the same gland.

The hyperplastic polypi are also of various forms. The gastric folds or rugae are enlarged, looking pressed together like the convolutions of the brain. The color varies according to the intensity of the process. In the acute and subacute state it is red and injected and may show hemorrhagic and purulent specks, or in more severe cases erosion, ulceration or necrosis may be seen. In the larger chronic edematous swellings the color is paler with mucus lying on or between the folds. The inflammatory hyperplasias have defective margins which merge with the normal mucosa. Their edges have a slow rise to a hilly slope, and they are flatter and more expansive on account of the inflammatory reaction. One does not receive the impression of an independent suddenly arising, isolated tumor. They are firm and rest immovable on the submucosa. Inflammatory polyps are rarely differentiated into head and stalk. On section the submucosa is seen to take no part in the growth as a connective tissue core, and the neighboring mucosa shows definite signs of chronic inflammation. The enlargement which forms the polyp is composed purely of mucosal proliferation of which the epithelial and fibrous components play a part. The epithelial cells lining the surface and those lining the crypts, including even the chief and parietal cells of the fundal mucosa, are replaced in areas large and small by intestinal type goblet cells and Paneth cells, this being a metaplasia or an acquired heteroplasia. There is also a relative increase in gland tissue which accounts for the over secretion of mucus, but the glands do not become so irregular and cystic as they do with an adenoma. The crypts contain a purulent mixture of mucus and leukocytes, which also lies on the surface. These hyperplastic areas are not

sharply delineated, but merge imperceptibly into the adjacent mucosa.

In some cases both the neoplastic and hyperplastic types may appear in the same specimen. At times the microscopic picture does not fit readily into the classification given, making it difficult or even impossible, to place the abnormality in a clear cut neoplastic or hyperplastic category.

MALIGNANT DEGENERATION

Although gastric polyposis is essentially a benign condition it is potentially capable of undergoing malignant degeneration. As stated succinctly by Kirklin and Broders, "Although polyps are at least primarily benign, they hold their benignant status precariously." Ewing has expressed the belief that all bukly gastric carcinomas may arise in benign polyps. It is the general consensus that the development of malignancy is precipitated by the following factors; the trauma caused by gastric peristalsis and the passage of coarse articles of food, the adenomatous nature of the benign lesions, and the chronic inflammation which may be pressent.

Malignant degeneration in gastric polyposis is similar to that occurring in epithelial tissue elsewhere. The usual changes are as follows; the epithelium becomes multi-layered, more undifferentiated, and exhibits polymorphism. The epithelium grows in an irregular fashion and mitotic figures become frequent. Finally the epithelium breaks through the muscularis mucosa and infiltrates the submucosa. It is only at this stage that one can be sure that the growth has assumed a malignant character.

The frequency of malignant change in gastric polyposis has been variously reported from 4.9 per cent to 41.2 per cent. The 12 per cent quoted by Brunn and Pearl is perhaps a figure which can generally be accepted. Regardless of the statistics reported, however, malignant degeneration should be suspected in all cases of gastric polyposis and the condition should be treated with this thought in mind.

SIGNS AND SYMPTOMS

The clinical picture presented by gastric polyposis is in no way characteristic. The signs and symptoms are extremely variable in both degree and duration. They vary according to the size, type, and location of the lesions. The condition may remain asymptomatic throughout life and only be found accidentally at necropsy. The duration of symptoms has been reported up to twenty-five years. There may be frequent remissions of varying duration, the longest reported being nine years. The returning symptoms are usually of a much greater severity than the original ones. The patient may subjectively experience vague abdominal distress, such as a dragging or a fullness in the epigastrium. He may have the sensation of slight pressure in the epigastrium, or that of severe pain. This pain has no distinct relationship to the taking of food, as the pain has been reported to come on before, during, and after meals. Some patients experience a marked anorexia, but the majority of patients have no changes in appetite. This is in direct contrast to those individuals having gastric carcinoma. The patient may complain of frequent eructations, nausea, and vomiting. They frequently notice that the vomitus is peculiar in that it resembles egg white very closely. This is due to the excessive mucus which the vomitus contains. Frequently the patient will notice gross blood in the vomitus, and upon occasion a small particle of a polyp or a whole polyp may be found. Occasionally the patient will notice that he has passed a polyp in the stool. The tearing loose of a polyp from its base is associated with severe pain, and a variable degree of hemorrhage, which may or may not give rise to hematemesis and melena. Depending on the amount of hemorrhage will be the degree of anemia and related weakness from which the patient will suffer. A polyp with a long pedicle may prolapse into the pylorus causing complete or partial obstruction. It may prolapse completely through the pylorus into the duodenum giving rise to a gastrogenous diarrhea. On physical examination one finds

little or nothing. One may notice evidences of an anemic pallor, and recent weight loss. Examination of the abdomen may reveal slight generalized rigidity, and mild to moderate tenderness in the epigastric region. In a very small percentage of the cases a palpable mass may be felt. This occurs most commonly in patients in whom the growth is large and located in the pyloric region.

The laboratory examination is extremely important in patients suspected of having this condition. The laboratory findings upon the examination of the gastric secretions are quiet significant. A large percentage of the patients (nine out of ten) will have an achlorhydria. One may also find associated with this that the gastric secretions are without digestive property and that they contain increased amounts of lactic acid. The egg white consistency of the secretions is frequently noted. Occasionally one may find particles of a polyp or a whole polyp in the gastric washings. Examination of the blood may reveal a secondary anemia depending on the amount of hemorrhage which has taken place. One may occasionally find a blood picture resembling that of pernicious anemia. Fecal specimens may be grossly bloody, and occasionally a polyp may be passed in the stool.

ROENTGEN AND GASTROSCOPIC EXAMINATION

One might think that the roentgen findings in gastric polyposis are pathognomonic of this condition, but this is not true. There are many times when even the most expert roentgenologists are hard pressed in differentiating gastric polyposis from gastric carcinoma. Gastric polyposis may present a roentgenographic picture so similar to gastric carcinoma that differentiation is impossible. However, the combined use of the roentgen ray and the gastroscope greatly increases the number of correct diagnoses, and a diagnosis of gastric polyposis should not be made until both roentgen and gastroscopic examinations have been done.

Before gastroscopic and, especially, roentgen examinations are carried out the stomach should be empty, for foreign bodies, such as food, excess mucus, and hair balls, may greatly confuse the picture. Thus, the patient should take no food or fluid after his evening meal preceding the morning of examination.

In the roentgen examination the barium mixture should be of such viscosity that it will coat the furrows of the tongue. The mixture should be given in small quantities —three to four teaspoonfuls at a time. The patient, who has been properly prepared, is screened for opacities. During the screening examination it may be noticed that peristalsis is unimpaired and that the stomach walls seem flexible. Overactive peristalsis will suggest obstruction. The tumor may be freely movable to gentle pressure; and, occasionally, a 'crater' appearance may be seen on a tumor, which appearance at the time of operation is proved to be an ulceration. The emptying time is usually normal. During this screening process 5 c.c. of fresh hydrogen peroxide may be given with three to four tablespoonfuls of barium—bubbles indicating blood, mucus, or other loose organic material.

After the screening process the patient is placed in the reclining position on a Potter-Bucky diaphragm, and trained to hold his breath on both inspiration and expiration. This is advantageous in that pressure can be made on parts otherwise covered by the ribs. Compression is used when the opaque media tends to pool in the stomach; otherwise, breathing movements and alterations in posture do what is needed. Films are taken with the patient sitting up, in the supine and prone positions, and also in the right and left oblique positions if desired. A funnel should be used to confine the radiation to the area which is being inspected.

Examination of the films will show the lesions, in the majority of instances, to be located on the greater curvature in the lower third of the stomach. The polyps project into the gastric lumen and produce ragged indentations in the barium shadow. These resemble mottled, fingerprint filling defects.

On gastroscopic examination, which is

excellent for the study of the gastric mucosa, one may see small reddened prominences separted by valleys of grayish mucosa. The prominences may contain many yellow ulcerations, and their surfaces may be irregular and nodular. The remaining gastric mucosa often appears normal.

It is important that the reader fully realizes that the differentiation of benign from malignant gastric tumors cannot be made on roentgen or gastroscopic examinations. Some roentgenologists claim that the defect in malignancy is annular, involving both curvatures, and, thus, can be differentiated from a benign lesion. Still others claim that infiltration of gastric musculature can be seen on the roentgenogram. Some gastroscopists claim to have the ability to make the differentiation between benign and malignant conditions by characteristics which are visible through the instrument, but not apparent in excised specimen or at autopsy. Until these premises can be definitely upheld by further study, all cases of gastric polyposis should be considered as malignant.

DIAGNOSIS

As has been seen from the above, the clinical picture alone is not characteristic enough to warrant a definite diagnosis of gastric polyposis. However, the following should suggest the condition to the clinician; an anemic patient who gives a history of vague abdominal distress and who further states that on several occasions he has vomited material looking much like egg white. He may or may not give the history of hematemesis and diarrhea. The finding of an achlorhydria upon the examination of the gastric secretions, along with excessive mucus, further substantiates the diagnosis. A mottled, fingerprint, filling defect in the lumen of the stomach seen on the roentgenogram and the direct visualization of multiple polyps on the gastric mucosa with the gastroscope is a positive confirmation of the diagnosis.

TREATMENT

Gastric polyposis has been treated in many ways with greater and lesser degrees of success. Douglas treated one patient by dropping a capsule of radium, tied to a string, into the stomach. This was left in the stomach for six hours with frequent changes in the position of the patient. Patient died three months later. Therapeutic roentgen ray treatment has been used with the relief of symptoms for variable periods of time. However, the symptoms return and are commonly increased in severity. It is impossible to tell whether the temporary relief of symptoms was due to the therapy or whether it was merely a spontaneous remission which frequently occurs. Cases of natural cure have been reported, in which torsion or strangulation of the pedicles have separated the polyps from their bases. It is generally accepted at present that the treatment of choice is surgery. Three procedures have been used; (1) local excision of the polyps through a gastrostomy; (2) gastric resection; and (3) gastric resection with the excision of a few remaining polyps in the gastric stump. Brunn and Pearl excised the polyps locally in four benign cases, achieving good results in only two of the four cases. They also carried out the same procedure in four malignant cases. Of these four, two patients were relieved for a period of five years, after which time there was a recurrence of symptoms. It can easily be seen, therefore, that local excision is grossly inadequate. The same men did gastric resections on five benign cases, and satisfactory results were obtained in all but one. The superiority of gastric resection over local excision is readily appreciated. Probably more important is the fact that gastric resection eliminates the possibility of malignant degeneration.

PROGNOSIS

Many cases of gastric polyposis remain benign and asymptomatic for years. However, all cases diagnosed should be treated, as soon as the patient is satisfactorily prepared, by gastric resection. If malignancy has not occurred, the prognosis is excellent following gastric resection. If malignancy has occurred, gastric resection is the most effective method of treatment. That all

gastric polypi are potentially malignant should be constantly kept in mind.

SUMMARY

A brief history of gastric polyposis has been outlined. The etiology, pathology, malignant changes, signs and symptoms, roentgen and gastroscopic examination, diagnosis, treatment, and prognosis have been discussed in detail.

CONCLUSIONS

Gastric polyposis is a rare condition occurring most frequently during the fourth and fifth decades in men more often than in women. There are two accepted theories of origin; the congenital and the inflammatory or acquired, the congenital theory being the more valid of the two. Two types of polyps occur; the polyadenome polypeaux, and the polyadenome en nappe, the former being hypertrophic in character, and the latter hyperplastic. The signs and symptoms are in no way pathognomonic of the condition, and the diagnosis should only be made after proper roentgen and gastroscopic examination. Since all cases are potentially malignant the treatment of choice is gastric resection. The prognosis is excellent in cases treated in this manner.

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WELL DESERVED HONORS

Elsewhere in the Journal is printed a list of the physicians of the state who have received from General Fleming of Selective Service a citation and a medal for the work on the draft boards that they did as civilians during the war. The medals and citations are well deserved rewards for the time and effort that were given voluntarily by a group of physicians to help the war effort. Many of these men for the entire duration of the war gave an untold number of hours to the examination of the man who was a candidate for induction into the army. At the beginning of the war a tremendous amount of time was contributed by most of these men to the examination of the young man who had been drafted. Hours and hours of purely voluntary effort were devoted to the tiresome and laborious routine examination of innumerable men. Later in the war the demands were not quite as strenuous but even then many doctors cheerfully and willingly gave of their time, extracted from the inordinate demands of wartime with a markedly reduced physician complement. The men to whom these medals have been awarded can feel proud to pass them on to their children and grandchildren to show that while they were in civil life they nevertheless helped make our war efforts successful.

It will be noted that many of the men to whom the medals have been awarded subsequently entered the armed services, and further gave of their services full time to their Country. It will be noted also that quite a few of the recipients of the medal have died. This illustrates well indeed the fact that it was the older man who was called upon for this particular service. We feel sure that having to take over the practice of other doctors, having to give time to this type of routine work, and having little time to care for themselves, was responsible for the death of some of the physicians who passed away during the war.

Incidentally the medals can be preserved and hung on the wall of the office by having them framed appropriately. The medals could well grace the office of doctors who so cheerfully, willingly and unselfishly gave their time to the type of work which at best could not be called exhilarating or stimulating.

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WAGNER-MURRAY-DINGELL BILLS

Numerous editorials have appeared in the Journal under the above caption and probably there will be more. It does seem more or less ridiculous to keep harping on one subject but when all is said and done this bill, or bills, are of fundamental and basic importance to the doctor. If the medical profession for one minute relaxes their

effort to defeat this particular legislation it is quite possible that it might be slipped over without their being aware of just what is going on at the moment. This, however, is doubtful but there is no doubt that we must be eternally vigilant.

At the present there is not very much likelihood of any immediate action being taken on these bills as Senator Murray has announced that until Congress reconvenes in the fall hearings on the bills will cease.

Comment has been made previously on the reporting of the hearings that are appearing in the Journal of the American Medical Association. If the busy practitioner has time to read the abstracted hearings presented before the Senate Committee on Education and Labor he will be struck by several features that well deserve comment. In the first place the opposition to the bills, led largely by the medical profession, seems to have so many arguments against the proposed legislation that are valid, accurate and well substantiated that it would seem as if the medical opponents of the bills have all the best of the argument. Of course the disinterested observer might say that this is the opinion of a physician but we feel sure that were the report of the hearings read by disinterested observers as for example, an English physician or a Norwegian doctor, or a Mexican barrister, or an Australian lawyer, they would agree with the statement that has just been made.

The proponents of the bills, on the other hand, seem to be very largely moved by sentiment. It is a very easy thing to appeal to sentiment, more particularly when it comes to such sentimental subjects as sick children. It is all right for such a charming and able physician as Dr. Martha Elliot to say that "either we spend money or we spend children." Does Dr. Elliot believe for a minute that the spending of very large sums of money is going to improve materially a situation in which maternal deaths, for example, are numbered only 24 to 10,000 mothers and the infant mortality of 40 deaths per 1,000 live births? Dr. Elliot has testified to the effect that every day the passage of the Wagner-Murray-Dingell bills is delayed is responsible for the death of large numbers of children. Again, we doubt very much if such a statement could be substantiated. We do not believe that many of these children would be saved even if they were treated by the professor of obstetrics at Johns Hopkins or Harvard University or by the professor of pediatrics at Columbia or Northwestern University. Statements such as Dr. Elliot has made are not convincing to the thoughtful individual although they do appeal to those who are hypersensitive and oversentimental.

Senator Murray, in his published comments on the bill that bears his name, states that one of the pressing reasons for this particular bill is because the indigent are dying without proper medical care. That is another statement that could be very well refuted. The indigent in large cities receive care which is comparable to that which may be given to a millionaire. Certainly in the rural sections, at least in Louisiana, the indigent are being treated at our big state hospitals by outstanding members of the medical profession. They receive medical care and attention which is notable. With good roads and the easy accessibility to even the most remote country districts it is possible to get a patient, for example, in a charity hospital within a very few hours. Furthermore the smaller state hospitals are wont to refer their difficult diagnostic and therapeutic cases to the two big state hospitals which, in equipment, lack nothing and whose medical personnel is highly skilled. If these revolutionary bills should be passed it is certainly not for the benefit of the extremely poor.

It is difficult indeed for any physician or for an intelligent layman to understand why medical services which are acknowledged to be the best in the world should suffer by the tremendous upheaval and disturbance in medical practice which the passage of the Wagner-Murray-Dingell bill is bound to occasion.

CORONARY ATHEROSCLEROSIS

Coronary disease is responsible for the death of more than one in five doctors in this country. Twice as many doctors die of coronary disease as die of cancer. Probably if studies were made or statistics kept in regard to the clergy, the law profession or to teachers the same general ratio would probably exist. The principal cause of death in men in this country at the period of their highest earing capacity is due to the pathologic involvement of a few grams of coronary intima. William Dock has made these statements in a recent paper in the Journal of the American Medical Association*. Dock speaks with authority. He is now professor of pathology at Cornell University Medical School and in the last several years has been professor of medicine at two prominent medical schools and professor of pathology at two other institutions. Many Louisiana physicians should have a vicarious interest in this author. His father some thirty-five years ago was professor of medicine at Tulane and is one who, despite the passage of eighty some years, still leads an active and vigorous existence.

The younger Dock has been interested for many years in coronary disease. He has recently called attention to the large number of young soldiers who die as a result of coronary atheroma while training in the United States. Many of these young men showed no evidence of arterial disease elsewhere. There are two predisposing factors of great importance in the etiology of atherosclerosis, to wit, hypertension and hypercholesteremia. In the younger man the latter factor is the more important of the two. The soldiers of the United States were the best fed soldiers in the world and their diet included butter, ice cream, milk, eggs; all substances high in cholesterol which might have played a factor in producing the cholesterol changes which were found in the coronary arteries of these young men. In people whose diet is cholesterol poor, but rich in whole grain, legumes and vegetable oils, coronary disease seems to be a rarity.

Probably one of the most remarkable features of coronary infarction or occlusion is the frequency with which it attacks men. It is only the occasional woman that suffers from this condition whereas, as noted before, it is extremely common in men. One of the important features of Dock's paper lies in the fact that he offers an explanation for the preponderance of males dying of coronary disease. This explanation is no hypothesis but is a definitely proved observation. Dock shows that, in the first place, the predilection of atherosclerosis of the coronary arteries is because they possess an intima which varies in thickness from ten to six hundred per cent of that of the media in the newborn infant. Secondly in the infant male whose intima was accurately measured, the average is 26 per cent whereas in the female infant it is only eight per cent.

Dock's paper is short, succinct and informative. It offers an explanation, in a very few pages, for two of the important problems of coronary disease which have not been previously answered: Why is the coronary artery more frequently involved than other relatively small arteries throughout the body and why atherosclerosis is so much more common in the male than in the other sex. Parenthetically it might be stated that Dock believes that the present appalling death rate in coronary disease may in the future become reduced as our understanding of hypertension and cholesterol metabolism becomes better understood. He even suggests the possibility that the incidence of coronary disease may fall to the insignificent level which is observed in the Chinese.

*Dock, William: Coronary atherosclerosis, J. A. M. A., 131:875, 1946.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1947 MEETING

Word has been received from the New Orleans Hotel Association that ample accommodations will be available for registrants at our meeting next year on May 12-14. Headquarters have not yet been selected for the meeting, however Dr. Edwin L. Zander has been appointed chairman of the Committee on Arrangements and important details will be handled promptly and ably under his guidance.

The Committee on Scientific Work, the special Committee to Consider Rearrangement of the Program and the chairmen of the various scientific sections will meet within the next few weeks to work out plans for a scientific program which will prove of interest and benefit to members attending the meeting.

STATE LEGISLATION

During the past session of the Louisiana Legislature two of the most important bills which the medical profession sponsored, after being passed by the senate and the house, were vetoed by the governor. Both of these bills called for additional expenditures which unquestionably influenced the veto owing to the fact that the legislature had appropriated money far beyond the state income. After fighting for several years for a state mental hospital close to New Orleans to take care of the tremendous number of patients at the other state institutions and after being successful in having the measure approved by both houses the appropriation of $300,000 was vetoed by the governor because a two-thirds majority did not prevail at the time action was taken.

Similar action befell the attempt to provide for needed statewide facilities for the chronically ill adjacent to our state hospitals. This bill was supposed to have had the endorsement of the administration and other powerful groups in addition to the medical profession, however after passing both houses it was finally vetoed by the governor for the same reason as for the mental hospital bill. Obviously owing to the exigencies of war there is an increased cost of maintenance, food, medical and special supplies for our present institutions which required increased appropriation for the already existing institutions. This increase had not been anticipated sufficiently to warrant the added expenditure of money far beyond the state income.

NATIONAL LEGISLATION

From a national viewpoint it will be interesting to note that from all information that we have received from accredited and reliable sources, Senate Bill 1606, known as the National Health Program, and featuring compulsory health insurance, will not be considered by the 79th Congress. You should know that it is regarded as a sound opinion that the satisfactory results obtained in this regard were due to the coordination and support of the medical profession of this country, and especially to the activities of our various state medical societies under the guidance of astute leaders in Washington and Chicago. These men represented the American Medical Association through the Council on Medical Service and Public Relations and through the National Physicians Committee. We certainly wish to commend them for the wonderful job which they have performed, not merely for the medical profession but for the public at large.

Apparently some members of the Committee on Education and Labor which has S. 1606 for consideration, felt that they would give some substantial support to this measure by stating in the press that they are in favor of some compulsory medical insurance, not controlled by the govern-

ment. However this is rather misleading and one will have to be influenced entirely by future action of this committee.

Please do not misunderstand the above statements as in anyway indicating that the question of federalization of medicine is a dead issue. Certainly in some form or fashion even if not considered by the 79th Congress it will be brought up in the 80th Congress after January 1, 1947.

There are certain things which have developed incident to these hearings before the Committee on Education and Labor. There was an attempt made by Senator Pepper on July 5 and July 15 to bring up before the Senate S. 1318, known as the Pepper Maternal and Infant Care bill. He failed in this attempt but undaunted he, along with Senator Taft who is also a member of the Committee on Education and Labor (and a reliable opponent to S. 1606) introduced in the Senate on July 15 a joint resolution No. 177 which was referred to the Committee on Finance. This joint resolution is an amendment to Title V of the Social Security Act of 1935, whereby $31,500,000 is appropriated instead of $5,873,000 appropriated in the original act for maternal and child health services. If this joint resolution prevails and an increased appropriation is granted it would, according to reliable critics, simply mean that we would go back to the year 1935 and consider all maternal and infant care services on a state basis, under the supervision and control of the state boards of health.

Senators who are members of the Committee on Education and Labor have sent us telegrams advising that action on S. 1318 has been postponed.

According to the action taken by our House of Delegates in May a communication was sent to every member of the State Society on May 30 advising that the agreement between our organization and the State Board of Health in regard to the EMIC plan, had been terminated, and asking that members of the State Society refuse to take further cases under this apprehension. Subsequent to this action the Executive Committee approved acceptance of cases where the patients were pregnant prior to May 8. Notice of this was carried in the organization section of the July issue of the Journal.

However, careful as we have tried to be in notifying our members of the above action, we are informed by the State Board of Health that some physicians are still making application to the Board of Health for remuneration for maternal and infant care under the old plan in existence during the war. Every member of the State Society is respectfully admonished not to accept any further cases under the EMIC program except as provided for by the Executive Committee, in order that the Board of Health may not be misled as to the desires of organized medicine in this state.

Recently, President Truman proposed extensive reorganization steps for the Executive Department of the federal government. Contained in his proposal was the removal of the Children's Bureau from the Department of Labor to the Federal Security Agency. Under existing statutes, the failure of either House of Congress to oppose this phase of the President's reorganization plan, the Children's Bureau was permitted to move into the Federal Security Agency.

It will be of interest to members of our organization to know that efforts are being made to have a uniform contract with the Veterans Administration throughout the state. This will enable veterans to be treated in their home towns by their own doctors and should prove to be a very satisfactory arrangement for the veteran and the physician.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

Aug. 14. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.

Aug. 15. Clinico-pathologic Conference, Touro Infirmary, 12 noon.

Aug. 22. DePaul Sanitarium Staff, 8 p. m.

Aug. 23. L. S. U. Faculty Club, 8 p. m.
Ochsner Clinic Staff, 8:30 p. m.

Aug. 26. Board of Directors, Orleans Parish Medical Society 8 p. m.

Aug. 28. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.

Aug. 29. Clinico-pathologic Conference, Touro Infirmary, 12 noon.

Aug. 30. New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.

Sept. 3. Orleans Parish Radiological Society, 7:30 p. m.

Sept. 5. Executive Committee, Baptist Hospital, 8 p. m.

EVERY TUESDAY, Medical Toastmasters, New Orleans Athletic Club, 6:30 p. m.

EVERY WEDNESDAY, Medical Discussions, Radio Station WDSU (1280 on your dial).

NEWS ITEMS

At the July meeting of the Society a motion picture entitled Scalp Avulsion was shown. At this meeting Dr. C. J. Vedrenne presented a paper on the Pathogenesis of Postoperative Atelectasis.

Dr. Donovan C. Browne attended the recent meetings of the American Gastro Enterological Association, and the American Gastroscopic Club at Atlantic City.

Dr. William B. Clark recently presented a preliminary report of his work on onchocerciasis in Guatemala before the Section of Opthalmology of the New York Academy of Medicine.

While in New York Dr. Clark took a two weeks' course in the surgery of keratoplasty under Dr. D. Townley Paton, director of the National Eye Bank, at the Manhattan Eye and Ear Infirmary, with the object in view of establishing a branch of the National Eye Bank in New Orleans.

Drs. Anna D. Colomb, Hunter P. Harris, Walter J. Otis and Walker Thompson attended the recent meeting of the American Psychiatric Association at Chicago.

Dr. Henry O. Colomb recently attended the meeting of the American College of Physicians at Philadelphia.

Dr. E. Carroll Faust was presented with a "Medal of Freedom". This award was given by the war department in recognition of his work on schistosomiasis in the Philippines.

Dr. Daniel J. Murphy recently addressed the Louisiana Pharmaceutical Association. The subject of his address was, "Medicine's Tribute to Pharmacy."

Dr. Murphy was appointed by Mayor deLesseps S. Morrison as a member of the Advisory Committee for Veterans' Affairs.

Dr. William A. Sodeman gave a series of six talks on internal medicine during the annual graduate short course for doctors in Jacksonville, June 17-22. The course was under the joint sponsorship of the University of Florida and the Florida state board of health.

Dr. Julius Lane Wilson attended the meeting of the National Tuberculosis Association at Buffalo, June 10-13.

The regular monthly meeting of the Journal Club of L. S. U.'s Department of Obstetrics and Gynecology was held June 5 at the Jung Hotel. Dinner was served first, after which several interesting papers on diabetes in pregnancy were given by members of the L. S. U. obstetric and gynecology resident staff of Charity Hospital. Members of the L. S. U. internal medical staff also contributed to the discussion. The meeting was attended by the visiting physicians, residents and interns of the L. S. U. obstetrics and gynecology unit of Charity Hospital. Guests included physicians from other services of the hospital and school.

NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

At a recent meeting of the New Orleans Graduate Medical Assembly the following physicians were elected to office:

Dr. William H. Gillentine, president; Dr. Joseph S. D'Antoni, president-elect; Dr. Randolph Lyons, first vice-president; Dr. H. Ashton Thomas, second vice-president; Dr. C. S. Holbrook, third vice-president; Dr. Max M. Green, secretary; Dr. Edwin H. Lawson, treasurer; Dr. C. Barrett Kennedy, director of program; Drs. J. O. Weilbaecher, Jr., and Ambrose H. Storck, assistant directors of program; Drs. L. C. Chamberlain, Frederick F. Boyce, Lucien A. Fortier, Howard R. Mahorner and Charles B. Odom to the executive committee.

The tenth annual meeting of the Assembly will be held February 24-27 with headquarters at the Municipal Auditorium.

WELCOME HOME

The following physicians were recently separated from military service: Dr. John J. Archinard, Ochsner Clinic; Dr. John G. Atwood; Dr. George G. Gill, Ochsner Clinic; Dr. John G. Menville, 606 Maison Blanche Bldg.; Dr. Carl N.

Wahl, 825 Maison Blanche Bldg.; Dr. Daniel W. Beacham, Charity Hospital; Dr. Michael N. Goldberg, Charity Hospital; Dr. Frederick L. Reuter, Charity Hospital; and Dr. John O. Redding, Jr., removed to Asheboro, N. C.

CALLED TO SERVICE

On June 10 Dr. J. William Rosenthal entered the medical corps of the United States Naval Reserve.

CORRECTION

In the last issue of the Journal we inadvertently listed Dr. E. L. Leckert, Jr., as having been called to military service as a *second* lieutenant. Dr. Leckert is a *first* lieutenant in the United States Army. We offer our apologies.

CHANGES OF ADDRESS

Dr. Irvin Cahen, 1441 Delachaise Street; Dr. John T. Crebbin, 1857 Benefit Street; Dr. G. Nelson Furbeck, Gante 1, Mexico, D. F. Mexico; Dr. John E. Garcia, 1305 Canal Building; Dr. W. Robyn Hardy, 601 Maison Blanche Building; Dr. Henry J. Lindner, Veterans Hospital, Lyons, N. J.; Malcolm F. Schwarzenbach, 1631 Murl Street; Lt. Jack R. Anderson, U. S. S. Howard Gilmore (AS-16), Fleet Post Office, New York; Lt. Thos. E. Furlow, U. S. Navy Recruiting Station, Dallas, Texas.

CHANGES IN MEMBERSHIP ROLLS

Four intern members were transferred to active membership—Drs. Wm. Fisher, Louis Levy, II, Robert M. Rose and Harry Rollings.

Two associate members were transferred to intern membership—Drs. Michael N. Goldberg and Frederick L. Reuter.

Transferred from active to inactive membership—Dr. James T. Nix.

Removed—Active members: Drs. Arthur J. Butt, Jr., Julius R. Fernandez, E. Borland Gill, John G. Pratt, Richard Selser, Miriam O. Vinnard, and Edgar Warren; associate members: Drs. Louis L. Friedman and John G. Hamilton; intern members: Drs. Howard M. Bordelon, Robert O. Harris, III, Frank S. Oser, Jr., O. L. Pollingue, Jr., John C. Taylor, Jr., Kendrick Taylor, Levon F. Wright and James H. Young.

RADIO PROGRAM

The following "Health Discussions" will be broadcast over radio station WDSU (1280 on your dial) during the month:

July 10th—Hypertension—Dr. Thomas Findley.

July 17th—Rabies in New Orleans—Dr. George H. Hauser.

July 24th—Polio—A Vacation Threat—Dr. Guy A. Caldwell.

July 31st—Infectious Diseases in Children—Dr. Frank Kinberger.

August 7th—Acute and Chronic Appendicitis—Dr. Daniel J. Murphy.

WELCOME HOME PARTY

On Monday, June 24 the Society officially welcomed home its members who served their country in World War II. A supper which was well attended, was held in the Ball Room of the New Orleans Athletic Club.

Dr. Daniel J. Murphy, president of the Society, delivered the following address of welcome: "Tonight our Society takes this opportunity to officially welcome home those of its members who so nobly answered the call of our country in the second World War. It is happy and proud to have upon its rolls so many names that have covered themselves with glory. As the president, it gives me so much satisfaction, in the name of the Orleans Parish Medical Society, to say *"Welcome Back"*. To me, no lengthy speech could possibly convey to you our pleasure in having all of you returned safe and sound to us. We are fortunate in the fact that our Society did not lose a single member as a war casualty. For this we are happy and are thankful. Fortunate as we are in not losing a member, some of the lustre of our happiness is dimmed when we think of the members of our Society who suffered the loss of some of their dear ones. To them our sympathy is extended and we hope that they can draw some measure of consolation in the thought that our nation is forever grateful to their departed ones for having made the supreme sacrifice so that all of us may live in peace. No flowery speech, no display of dramatics, no eloquence in oratory can possibly express to you our joy in having all of you returned to us. So in the name of the Society, I know of no better way to show our appreciation to you for what you have done for us and the pleasure we have in your safe return than by saying *"Welcome back and thank God that you are back again with us to stay!"*

Dr. Edmond Faust responded for the Army; Dr. Shirley C. Lyons for the Navy; Dr. John Oakley for the Marines; Dr. Samuel Colvin for the Tulane Hospital Unit; Dr. Charles Miangolarra for the L. S. U. Hospital Unit.

Between each response the theme song of that branch of service was sung; accompaniment at the piano was by Dr. Emile A. Bertucci.

The meeting closed with the singing of the Star Spangled Banner.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SELECTIVE SERVICE AWARDS

Doctors serving on Medical Advisory Boards in Louisiana who have received the Selective Service Medal:

Drs. Maurice Campagna, Ernest C. Samuel, Julius Wilson, G. C. Anderson, H. F. Brewster, Edmund Connely, Val H. Fuchs, Edgar Hull, S. C. Jamison, D. Urban Maes, Leon J. Menville, J. R. Schenken, H. T. Simon, B. J. DeLaureal, W. H. Harris, C. S. Holbrook, F. E. LeJeune, Randolph Lyons, E. Alton Oschner, J. T. O'Ferrall, Victor C. Smith, T. A. Watters, E. B. Vickery, Charles A. Bahn, L. A. Fortier, Lewis Golden (Deseased), Roy B. Harrison, Sam Hobson, E. H. Lawson, J. H. Musser, H. W. E. Walther (Deseased), all of New Orleans.

Drs. Dean H. Duncan, H. A. Durham, L. W. Gorton, W. R. Harwell, Joe E. Heard, Douglas L. Kerlin, W. Sam Kerlin, Morgan W. Matthews, W. R. Mathews, I. B. Rougon, John L. Scales, all of Shreveport.

Drs. A. J. Comeaux, Lafayette; L. O. Clark, Lafayette; C. C. DeGravelles, New Iberia; C. E. Hamilton, Lafayette; Paul H. Kurzweg, Jr., Lafayette; L. B. Long, Lafayette; Jos. D. Martin (Deceased), New Iberia; John M. Miles, Lafayette; J. D. Frazar, DeRidder.

Drs. S. F. Hatchette, Louis A. Hebert, R. G Holcombe, D. C. Iles, G. C. McKinney, O. W. Moss, and T. H. Watkins, all of Lake Charles; J. Q. Graves, C. P. Gray, W. M. Hunter, C. P. Jarrell, D. M. Moore, P. L. Perot, and John Pracher, all of Monroe; H. O. Barker, M. H. Foster, M. S Freiman, O. B. Owens, P. K. Rand, N. T. Simmonds, and B. M. Wilson, all of Alexandria; William B. Chamberlin, U. S. Hargrove, Brinsfield King, L. F. Lorio, T. Jeff McHugh, T. H. Pargen, Lester J. Williams, and Howard B. Witter, all of Baton Rouge.

To the following were also awarded medals and certificates:

Ascension Parish: Drs. Forest E. Baker, Gonzales; Henry A. Folse, Donaldsonville; Gerald L. Gaudin, Gonzales; Earl A. Schexnayder, Donaldsonville.

East Baton Rouge Parish: Drs. W. L. Comeaux, Oswald W. Cosby, W. A. Dial, W. R. Eidson, Frank W. Pickell, Felix Boizelle, J. P. Griffon, J. R. Godfrey, H. C. Hatcher, W. K. Irwin, Rhett G. McMahon, J. D. Martin, Jr., H. Guy Riche, Ashton Robins, Charles H. Voss, D. D. Barnett, W. H. Cook, F. U. Darby, L. J. Kern, Jr., H. W. A. Lee, Jos. J. Noto, Philip B. Polito, William McHugh Simmons, W. B. Singletary, W. Shewen Slaughter, J. A. Tucker (Deceased), B. E. Nelkin, and C. O. Bingham, all of Baton Rouge.

East Feliciana Parish: Dr. C. S. Toler, Clinton.

Iberville Parish:Drs. William E. Barker, Jr., J. Cyril Eby, Adrian Landry, and Eugene Holloway, all of Plaquemine.

Rapides Parish: Drs. R. O. C. Green, S. L. Calhoun, J. T. Cappel, A. L. Culpepper, J. P. Durham, H. P. Forsyth, H. H. Hardy, Ralph Lampert, L. F. Magruder, R. G. Masterson, R. E. C. Miller, W. M. McBride, R. U. Parrott, G. W. Smith, R. B. Wallace, Harry Koretsky, James W. Welch, M. B. Pearce, N. M. Brian, Jr., V. F. Chicola, J. H. Landrum, R. E. McGill, W. L. Murrell, and E. R. Pincus, all of Alexandria. Dr. B. F. Bremer, Glenmore. Dr. R. C. Scott, Lecompte.

Caldwell Parish: Drs. E. L. Carroll and D. O. Sherman, Columbia. Dr. J. G. Norris, Farmerville.

East Carroll Parish: Drs. D. F. Davis, and J. Preston Davis, Lake Providence.

Franklin Parish: Drs. L. F. Robinson, and J. D. Rogers, Winnsboro.

Jackson Parish: Drs. T. A. Dekle, M. M. Hearne, and C. S. McDonald, Jonesboro. Dr. George H. Robinson, Hodge.

Catahoula Parish: Dr. P. W. Calliham, Harrisonburg.

Concordia Parish: Drs. A. J. Hodges, and Joseph H. Pankey, Ferriday.

Grant Parish: Dr. Nathaniel M. Brian, Montgomery. Dr. D. V. Donaldson, and J. F. Woodall (Deceased) Colfax. Dr. J. W. Scott, Rochelle.

Lasalle Parish: Dr. E. L. Miller, Jena. Dr. F. A. Thomas, Urania. Dr. David Trax, Trout.

Natchitoches Parish: Drs. R. E. Corkern, W. M. Knipmeyer, M. H. Phelps, W. H. Pierson, R. S. Roy, all of Natchitoches.

Vernon Parish: Dr. E. E. Archibald, Alco. Dr. M. S. Stephens, Hornbeck. Drs. W. E. Reid, E. M. Shaw, and M. W. Talbot, Leesville.

Winn Parish: Drs. J. F. Faith, S. C. Fittz (Deceased), and J. T. Mosley, all of Winnfield.

Livingston Parish: Drs. A. A. Murray, and Montgomery Williams, Denham Springs.

Pointe Coupee Parish: Drs. E. G. Durel, J. M. Mosely, J. W. Plauche, Jr., and F. F. Rougon, all of New Roads.

St. Helena Parish: Dr. William E. Day, Greensburg.

Tangipahoa Parish: Drs. A. L. Lewis, and M. B. Small, Amite. Dr. Luther L. Ricks, Independence. Drs. R. G. Korniker, and G. T. Scott, Jr., Ponchatoula. Drs. Alexander J. Feder, C. D. Overton, M. C. Wigington, Carlos D. Alessi, Collins P. Lipscomb, and John D. Thomas, all of Hammond.

West Baton Rouge Parish: Drs. D. W. Landess, and Paul B. Landry, Port Allen.

West Feliciana Parish: Drs. Cecil C. Blakeney, and Philip A. Niebergall, St. Francisville.

Avoyelles Parish: Drs. A. B. Cairns, and H. C. Jones, Bunkie. Dr. J. C. Goldner, Marksville. Dr. Philip Jeansonne, Plaucheville. Dr. Kirby A. Roy, Mansura.

Lincoln Parish: Drs. Marvin T. Green, J. B. Harris, S. L. White, and E. J. Young, Ruston. Dr. Jos. L. Smith, Dubach.

Madison Parish: Drs. D. H. Allen, F. A. DeJean, E. Otis Edgerton, G. W. Gaines (Deceased), Howard Clay Sevier (Deceased), and William L. Yerger (Deceased), all of Tallulah.

Morehouse Parish: Drs. William V. Garnier, John Newton Jones, Rae Byrd Leavell, W. A. Rodgers, and Smith I. Sims, all of Bastrop. Dr. E. L. Maddry, Oak Rridge. Guy D. Williams, Mer Rouge.

Ouachita Parish: Drs. H. V. Collins, Henson Coon, A. E. Fisher, J. T. French, Henry E. Guerriero, C. H. Hill, C. U. Johnson, W. E. Jones, John G. Snelling, Jr., George Varino, Irving J. Wolff, Glenn T. Gallaspy, and Ellis Powell, all of Monroe.

Richland Parish: Drs. John C. Calhoun, and H. C. Chambers, Rayville, Drs. Sheldon Teer, and C. C. Thompson, Delhi.

Tensas Parish: Dr. Joseph Whitaker, St. Joseph.

Union Parish: Dr. C. C. Colvin, Bernice. Drs. C. C. Francis, and W. P. Lambeth, Farmerville.

West Carroll Parish: Dr. Daniel W. Kelley, Oak Grove.

Allen Parish: Drs. Gurdon Buck, Kinder. Dr. E. H. Kent, Oberlin. Dr. A. D. Mangham, Clarks. Drs. L. F. Gray, M. V. Hargrove, and W. Rigsby Hargrove, all of Oakdale.

Beauregard Parish: Drs. Luke Marcello, S. T. Roberts, and Thomas R. Sartor, all of DeRidder.

Calcasieu Parish: Drs. B. D. Atchley, T. B. DeLaurel, Willie G. Fisher, Ben Goldsmith, Fritz LaCcur, Walter O. Moss, L. L. DiGiglia, R. P. Kandle, J. G. E. Barham, all of Lake Charles. Dr. A. H. Forman, Vinton. Dr. George S. Lyons, DeQuincy. Drs. Robert B. Marshall, and W. A. K. Seale, Sulphur.

Cameron Parish: Dr. J. B. Colligan, Hackberry.

Jefferson Davis Parish: Drs. Trois E. Johnson, Richard S. Kramer, Louis E. Shirley, Morgan Smith, F. W. Harrell, all of Jennings. Drs. Claude Martin, and John G. McClure, both of Welsh.

Acadia Parish: Dr. C. D. Brunt, Iota. Drs. J. W. Faulk, H. L. Gardiner, S. R. Henry, A. R. Morgan, E. S. Peterman, all of Crowley. Drs. John S. Baily, and T. T. Tolson, both of Church Point. Drs. Ernest C. Faulk, and R. C. Webb (Deceased), both of Rayne.

Evangeline Parish: Dr. R. E. Dupre, Ville Platte. Dr. O. L. Freeman, Basile. Dr. Frank Savoy, Mamou. Dr. Arthur Vidrine, Ville Platte, and Dr. G. G. Fontenot (Deceased), Ville Platte.

Iberia Parish: Drs. L. A. Breffeilh, Lewis B. Crawford, Orien E. Dalton, Henry J. Dauterive, Harold M. Flory, Edwin L. Landry, and Wilton P. Tilly, all of New Iberia. Dr. Wilfred Morgan, Weeks. Drs. C. W. Phillips, and Lastie M. Villien, both of Jeanerette.

Lafayette Parish: Drs. J. O. Duhon, Clarence J. Saloom, and C. K. Olivier, all of Lafayette.

St. Landry Parish: Drs. Lionel J. Bienvenu, W. R. Lastrapes, S. J. Rozas, Oscar J. Bienvenu, Ertemon Lafleur, all of Opelousas. Dr. George A. Azar, Washington. Dr. Leo L. Collins, Port Barre. Drs. Harry Jenkins, Charles W. Lewis, and Weston R. Miller (Deceased), all of Eunice. Dr. Gordon Morgan, Melville. Dr. Dewitt Grover, Rackley.

St. Martin Parish: Drs. J. L. Beyt, A. R. Corne, and P. H. Fleming (Deceased), all of St. Martinville. Drs. L. A. Morrogh, and S. D. Yongue, both of Breaux Bridge.

St. Mary Parish:Dr. J. C. Berwick, Berwick. Drs. T. H. Gueymard, and F. H. Metz, both of Morgan City. Dr. J. R. Rushing, Baldwin. Drs. A. C. Kappel (Deceased), L. J. Supple, and G. Vasquez, all of Franklin.

Vermilion Parish: Dr. L. M. Boudreaux, Erath. Dr. George L. Gardiner, Sr., Gueydan. Drs. Thomas Latiolais, Edward R. Villemez, and Leo Saporito (Deceased), all of Kaplan. Drs. J. E. McClellan, Shelley Mouledous, Marion A. Young, Jr., and L. E. Chauvin, all of Abbeville.

Bienville Parish:Dr. V. W. Fletcher, Ringgold. Drs. Orum L. Kidd (Deceased), and Carl L. Langford, both of Gibsland. Dr. J. L. Wade, Arcadia.

Bossier Parish: Drs. H. N. Barnett, and J. B. Hall, Jr., both of Benton. Dr. D. C. McCuller, Bossier City.

Caddo Parish: Drs. W. B. Allums, Dorf Bean, Thomas V. Bush, F. A. Buvens, L. H. Pirkle, E. L. Sanderson (Deceased), Thomas R. Simpson, R. R. Wilson, Wm. M. Hall, G. H. Cassity, I. F. Hawkins, C. H. Potts, H. J. Quinn, W. J. Sandidge, Ernest B. Flake, W. B. Heidorn, Clifford R. Mays, T. J.

Smith, J. Dudley Talbot, J. M. Bodenheimer, and R. B. Delee, all of Shreveport. Dr. Matthias M. Collins (Deceased), Hosston. Drs. J. W. Mobley, and C. H. Pardue, both of Vivian. Dr. William S. Siler, Jr., Ida. Dr. T. B. Tooke, Belcher.

Claiborne Parish: Drs. E. A. Campbell, and B. L. Stinson, both of Homer. Drs. A. G. Worley, and W. L. Waller, both of Haynesville.

Desoto Parish: Drs. W. B. Hewitt, and R. A. Tharp, both of Mansfield.

Red River Parish: Dr. Jack Carlisle, East Point. Dr. L. S. Huckabay, Harmon. Dr. W. B. Hunter, Coushatta.

Sabine Parish: Drs. S. F. Fraser, J. K. Phares, and Gustave F. Weber, all of Many. Dr. R. L. Parrott, Zwolle. Dr. H. M. Prothro, Pleasant Hill.

Webster Parish: Drs. Claude M. Baker, J. B. Benton, S. F. Martin, and R. E. Smith, all of Minden. Dr. W. C. Gray, Springhill. Dr. John Pugh, Cotton Valley.

Assumption Parish : Dr. Dewey Billeaudeaux, Klotzville. Dr. Julius W. Daigle, Belle Rose. Dr. Henry A. LeBlanc, Paincourtville. Drs. Nelson A. Cox, W. E. Kittredge (Deceased), William Pugh, and C. S. Roger, all of Napoleonville.

Jefferson Parish: Drs. John W. Atkinson, Carroll F. Gelbke, Charles Gelbke, and Floyd M. Hindelang, all of Gretna. Dr. Philip P. LaBruyere, Marrero. Dr. Joseph J. Massony, Westwego. Dr. L. J. Genella (Deceased), Kenner. Dr. J. S. George, Metairie.

Lafourche Parish: Drs. Charles J. Barker, F. T. Beatrous, J. L. Danos, and Murphy P. Martin, all of Thibodaux.

Plaquemines Parish: Dr. Benj. H. Carlton, Port Sulphur. Dr. B. R. Slater, Belle Chasse.

St. Bernard Parish: Drs. A. V. Filizola, Felix Planche, N. P. Trist, Jr., all of Arabi.

St. Charles Parish: Drs. Samuel L. Gill, and Roy A. Kelly, both of Destrehan. P. T. Landry, Norco.

St. James Parish: Dr. Stephen R. Campbell, Hymel. Dr. Anthony J. Nobile, Lutcher. Dr. Harry J. Schmidt, Convent. Dr. L. O. Waguespack, Vacherie.

St. John Parish: Drs. Pierre A. Donaldson, and J. S. Parker, both of Reserve. Dr. J. R. Fernandez, Wallace. Drs. Remy F. Gross, and W. F. Guillotte, Laplace

St. Tammany Parish: Drs. H. E. Cannon, and H. E. Gautreaux, both of Covington. Dr. F. R. Singleton, Slidell.

Terrebonne Parish: Drs. S. Clark Collins, J. B. Duval, S. Ernest Ellender, W. A. Ellender, H. L. Haydel, M. F. Houston, and H. P. St. Martin, all of Houma.

Washington Parish: Drs. J. S. Desporte, W. A. Fleming, W. S. Harrell, E. E. Lafferty, Frank Mock, and A. G. Murphy, all of Bogalusa. Drs. W. R. McGehee, Jr., and H. A. Stafford, both of Franklinton.

Orleans Parish: Drs. C. J. Brown, Philip Montelepre, Robert A. Robinson, Jr., Samuel A. Romano, C. J. Tripoli, Shirley C. Lyons, Manuel Gardberg, Howard R. Mahorner, George C. Miramon, William J. Rein, I. L. Robbins, John J. Archinard, Edward Matthews, Murrell Kaplan, Philip A. Boudreaux, James C. Cole, J. Holmes Smith (Deceased), W. Norton Voorhies, L. A. Monte, C. F. Bellone, Frank F. Gambino, Jr., W. G. Troescher, Jos. J. Ciolino, Lucien Delery, Edward B. Faget, Philip H. Jones, Jr., Benj. L. Newell, T. T. Batson, Hugh T. Beacham, Frank Chetta, F. C. Hava, David Hyman, J. Henry Larose, Jr., Lewis H. Levy, Warren L. Rosen, C. J. Wichser, Owen F. Agee, Louis L. Friedman, T. A. Jung, D. V. Longo, George A. Cronan, Joseph M. Hountha, T. F. Kirn, Joseph P. Palermo, Claude V. Perrier, Joel B. Gray, John Morgan Lyons, Harold A. Bloom, M. L. Michel, Rawley Penick, Jr., J. D. Russ, Willard Wirth, Woodward D. Beacham, Frank L. Cato, Jr., B. G. Efron, E. R. Guidrey, R. T. Liles, James L. Locascio, Frank L. Loria, Spencer B. McNair, Charles S. Healey, Peter Everett, Jr., H. B. Alsobrook, C. Gordon Johnson, J. W. A. Smith, Charles L. Bloom, Hymen Cohen, Lucien H. Landry, Alberto Prieto, J. D. Rives, A. F. Brock, P. J. Saleeby, E. A. Bertucci, R. S. Crichlow (Deceased), Esmond A. Fatter, F. Temple Brown, J. M. Davidson, B. Hochfelder, and J. M. Perret, all of New Orleans.

TREUTING NEW HEAD OF STATE BOARD OF HEALTH

Dr. Waldo Louis Treuting has been appointed to succeed Dr. David E. Brown as president of the State Board of Health. Dr. Treuting is a native of New Orleans and graduated from Tulane Medical School in 1934. After a year's internship at Charity Hospital he was appointed to the State Board of Health. For three years he was head of the division of epidemiology and preventive medicine. While serving the Board of Health he received his degree of master of public health in 1939 from Johns Hopkins University. His progress in the State Board of Health has been steadily upward and during the war he was, next to Dr. Brown, the highest ranking officer in the department.

Dr. Treuting is well trained for public health work. He should make an excellent director of the department. Governor Davis is to be congratulated on appointing a man who has made public health his life work and who is qualified in every respect as a well trained public health officer.

NEWS ITEMS

Dr. Howard Mahorner, assistant professor of clinical surgery, Tulane, addressed the Mobile Civic Forum recently on the subject of Socialized Medicine — the Phantom Approaches.

It has been announced by President Rufus C. Harris, of Tulane, that a grant of $35,000 for the

purpose of studying the effect of climate on the circulatory system has been made to the university by the Life Insurance Medical Research Fund. This research work will be directed by Dr. George E. Burch, associate professor of clinical and experimental medicine.

A breakfast was given to the past vice-presidents of the American Medical Association during the recent meeting of the organization in San Francisco. Dr. L. H. South, of Louisville, and Dr. J. Watts Stovall, president of the Kentucky State Medical Association, were hosts. There are 21 surviving vice-presidents of the A. M. A. of whom two, Dr. Rudolph Matas and Dr. John H. Musser, reside in New Orleans.

Drs. George E. Burch, Travis Winsor and L. LeRoy Kimball, were one of three groups awarded a certificate of merit at the scientific exhibit of the A. M. A. The award is based on the originality of the investigation and the excellence of presentation.

At the quarterly meeting of the New Orleans chapters the Board of Directors of the Red Cross appointed the following medical advisory committee: Dr. Joseph A. Danna, Chairman, Dr. Val H. Fuchs and Dr. C. Walter Mattingly.

President W. B. Hatcher of the Louisiana State University announces an addition of 40 new faculty members to the university medical school plus 23 more members added to the staff of those who have returned from duty in military service since December 1.

Honorary memberships in the Brazilian Academy of Military Medicine were recently bestowed upon Major General Norman T. Kirk, The Surgeon General, and Colonel Leon L. Gardner, MC, Director of Army Medical Library.

INTERNATIONAL COLLEGE OF SURGEONS

The executive secretary of the International College of Surgeons announces that 17 operative surgical clinics will be held at the Detroit Assembly October 21-23. The convocation address will be delivered by Ex-Ambassador to Japan, Joseph C. Grew.

AMERICAN COLLEGE OF PHYSICIANS

The 28th annual session of this important organization will be held in Chicago April 28-May 2, 1947. Dr. David P. Barr, President of the College, is in charge of the program of general sessions and lectures. Dr. LeRoy H. Sloan is general chairman in charge of the program of hospital clinics and panels as well as of local arrangements.

SOUTHERN MEDICAL ASSOCIATION

According to the notice sent out by Mr. C. P. Loranz, Secretary-Manager of the Southern Medical Association, the Miami meeting of this organization will open at noon Monday, November 4 and will continue through Thursday afternoon, November 7. The general headquarters will be at the Municipal Auditorium in Bayfront Park. In this notice sent out by the secretary-manager, the various hotels are listed. It is suggested concerning hotel space that where the hotel of choice can not accommodate the applicant, reservations be made through the hotel committee.

After-meeting trips are being arranged for those who wish to go to Havana and/or Nassau.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

The National Society for the Prevention of Blindness will hold a three-day conference November 25-27 at the Hotel Pennsylvania, New York.

ARMY CONSULTANTS

The surgeon general of the army has announced that three New Orleans physicians are amongst the 74 civilian doctors appointed as consultants to the secretary of war. The three appointees are Dr. Champ Lyons, surgical consultant, Dr. Michael E. DeBakey, consultant in general surgery and military history consultant, both of the surgical department of the Tulane Medical School and Dr. Roy H. Turner, of the Tulane Department of Medicine, medical consultant.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending June 1 showed the following diseases in numbers greater than ten: Unclassified pneumonia 126, cancer 127, measles 106, pneumococcic pneumonia 59, pulmonary tuberculosis 32, amebiasis 24, malaria 24, mumps and rheumatic fever 18 each, scarlet fever 13, septic sore throat 12, influenza 11 and typhoid fever 10. There were three cases of poliomyelitis reported this week, two from Livingston and one from Iberville Parish. The number of pneumonias, unclassified and pneumococcic, is very remarkable for this time of the year. At the same time that the weekly morbidity report was sent out the monthly morbidity report for venereal diseases was distributed. For the month that ended May 31 there were listed 1971 cases of syphilis, 1710 of gonorrhea, 66 of chancroid and 20 of granuloma inguinale.

There was a truly remarkable fall in the number of reportable diseases for the week which closed June 8. The only disease which occurred in figures greater than ten was measles with 34 cases listed. However it can be noted that there were nine cases of poliomyelitis distributed throughout the state,

no one parish having more than two cases. For the week which came to a close on June 15 there were sent to the State Board of Health reports of 117 cases of measles, 62 of pulmonary tuberculosis, 26 of malaria, 22 of mumps, 17 of septic sore throat, 13 of whooping cough and 11 of meningococcus meningitis. Only three cases of poliomyelitis were reported this week. For the week ending June 22 for the first time in many a week pulmonary tuberculosis led all other reportable diseases with 59 incidences followed by 55 of measles, 27 of mumps, 18 of whooping cough, 14 of septic sore throat and 13 of typhus fever. There were eight cases of poliomyelitis listed for this week, four of which originated in Orleans Parish; the others scattered throughout the state. So far this year there have been listed 58 cases of poliomyelitis, a figure well above that of last year.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce reported that for the week ending June 15 there were 103 deaths in the City of New Orleans. These deaths were divided 71 white and 32 colored with seven of the deaths occurring in children under one year of age. This was a very marked reduction from the previous three year average. There was a fairly large increase in the number of deaths for the week which terminated June 22. Of the 137 deaths in New Orleans this week 89 were white, 48 colored and 12 infants under one year of age. The deaths had materially fallen in number for the week which came to a close June 29. The three year average for the corresponding week has been 160 but in this week only 115 people died in the city, of which 76 were white, 39 colored and five infants. The astounding low figures were maintained for the week which closed July 6. The 106 people who died in New Orleans were divided 67 white, 39 colored and 11 infants. The low mortality rate for the City of New Orleans for the past year has been truly remarkable.

LOUISIANA STATE BOARD OF MEDICAL EXAMINERS

Governor Davis has announced the reappointment of Dr. Roy B. Harrison, Dr. Leon J. Menville and Dr. Rhett McMahon to the State Board of Medical Examiners. These reappointments were made on recommendation of the House of Delegates of the Louisiana State Medical Society. These three men have proved their efficiency and their ability properly to carry out this somewhat difficult assignment.

CARROLL D. OVERTON
1902 - 1946

Dr. Carroll D. Overton, of Hammond, was shot and killed on July 1 by a former patient who then committed suicide. A tragedy such as this is one of the occupational hazards of medicine. Time and again doctors are murdered by insane patients who believe that their illness is dependent upon improper treatment.

Dr. Overton was a graduate of Tulane University Medical School and practiced medicine in Hammond after completing his internship.

BERT LANDON STINSON
1870 - 1946

Dr. Bert Landon Stinson, of Homer, died on July 8 of hemorrhage of the descending aorta. He was a graduate of Rush Medical College, 1900 and interned at Milwaukee Hospital, Milwaukee, Wisconsin in 1901. In 1927 he came to Amite, Louisiana, serving as Director of the Tangipahoa Parish Health Unit. From Amite he moved to New Iberia and in 1939 became Director of the Claiborne Parish Health Unit, which position he held until the time of his death.

WOMAN'S AUXILIARY

The Woman's Auxiliary to the Tangipahoa Parish Medical Society met with Mrs. DeLoach Thames on Thursday, July 11, for their regular monthly meeting, Mrs. Glen Scott, President, presiding. For the Program, reports were given on the state convention, recently held in Alexandria. A complete report was given of all activities by those who attended, Mrs. Glen Scott, Mrs. DeLoach Thames, and Mrs. M. C. Wiginton. Mrs. Wiginton gave a brief resumé of the report of the past president, Mrs. Paul Lacroix. These reports created so much enthusiasm among members that all decided they could not afford to miss the next state meeting.

Following the business session Mrs. Thames served very delightful refreshments.

In spite of the heat this was a most interesting meeting and it was very gratifying to have wives of the medical profession so congenial and enjoy such rich fellowship as was evident.

Two new members were added to the roll, Mrs. S. J. Dileo of Independence, and Mrs. R. H. Alldredge, associate member. Others present were Mrs. Glen T. Scott and Mrs. Collins Lipscomb of Ponchatoula, Mrs. S. J. Cali, Mrs. C. D. Alessi, Mrs. M. C. Wiginton.

Other auxiliaries disband during the summer months, but Tangipahoa Parish, so newly organized, thought it wise to have regular meetings, and although these meetings will be purely social they promise to be very entertaining and interesting. In the fall when other auxiliaries begin to function, Tangipahoa Parish Auxiliary will begin regular activities.

BOOK REVIEWS

Normal Lives for the Disabled: By Edna Yost and Lillian M. Gilbreth. New York: Macmillan Company, 1945. Pp. 298. Price, $2.50.

This admirable volume is published especially for those who are mentally or physically impaired because, with relatively few exceptions, handicapped persons can attain economic independence and a normal or satisfactory social situation. It tells them how to go about it and is most helpful for their families and friends — for everyone who wants intelligently to take part in helping the disabled achieve useful, happy lives.

Every handicapped individual-civilian or veteran can and should initiate reconstructive thinking and efforts to help himself as early as possible. From years of experience, the authors know that everyone, when real need arises, has a superiority in one line or another which can help compensate for physical or psychologic loss even when the defect is a severe one. They have seen countless men and women — not just those with unusual talents, gifts or skills — conquer their defects, find suitable places in business or industry, and enjoy themselves, their homes and their friends.

Here is a comprehensive, practical and intelligently presented survey of what has been and is now being done to help impaired persons rehabilitate themselves. It reflects the views and experiences of specialized teachers, industrialists, psychiatrists and psychologists, as well as of physically or psychologically injured men and women themselves who have successfully solved their difficulties. It also contains valuable material on vocational problems as they must be met by the variants, and excellent descriptions of these men and women in an amazing variety of jobs in the United States today.

Though small in size this manual possesses qualities and values which are superlative. Its service offerings and standards are high. Its material is designed to keep pace with new human needs and demands. To reiterate, it engagingly expresses the authors' convictions of the self-respect and happiness which handicapped men and women can attain.

C. P. MAY, M. D.

The Intervertebral Disc: With Special Reference to Rupture of the Annulus Fibrosus with Herniation of the Nucleus Pulposus: By F. Keith Bradford, M. D. and R. Glen Spurling, M. D. Springfield, Ill., Charles C. Thomas, 1945. Pp. 192, 70 illus. Price $4.00.

This second edition of a truly fine monograph brings one up to date on the important subject of the intervertebral disc. Importance is stressed in the early part of the book on the fundamental anatomy, physiology and pathology of the subject. The relation of these basic facts and their correlation with the clinical syndrome is adequately expressed in the second portion of the book.

The localization and differential diagnosis of the ruptured annulus fibrosis is made clear by means of tables and drawings. The procedure following a diagnosis of first following conservative measures, and only resorting to surgical removal of the disc when these fail is sound medicine in a comparatively new syndrome. The authors do not state which spines should be fused, although they discuss the fact that this procedure should be followed in certain cases. However, in their case reports no spines are fused.

Although 13 case reports are given, only one case of mistaken diagnosis is mentioned. In the differential diagnosis little attention is given to mistakes in diagnosis.

This monograph is adequately indexed, profusely and well illustrated, and is recommended for its research, clarity and context to all physicians whose patients complain of persistent backache.

FRED S. MARSHALL, M. D.

Injury and Death Under Workmen's Compensation Laws: By Samuel B. Horowitz. Boston, Wright & Potter Printing Co., 1944. Pp. 846. Price $6.00.

Compensation acts in the United States are now of age. All over the country there are thousands of men and women injured every year in industries and existing acts are a boon to them and their dependents. Annually 2,000,000 workers become casualties; 18,000 die; 100,000 are maimed for life; and the rest recover after varying periods of time. About $200,000,000 a year reach the hands of workers, their dependents, and those who furnish medical and hospital care.

Although the basic theory and principles of workmen's compensation are relatively simple, their practical application involves a tremendous number and variety of important administrative details and requires a high degree of talent and alert, diligent effort. And, too, compensation laws are put on the books for the benefit of workers, not employers or insurance companies.

In the sections on Personal Injuries and By Accident, there are many important observations of which the following are interesting examples: (1) Nervous shock causing incapacity for work is as much a personal injury by accident as a broken limb or other physical injury. When you have that which requires treatment of the body, it means that a portion of the body (visible or invisible does not matter) is in a state of ill health. (2) Traumatic neuroses following physical injuries are almost universally compensated, even

though financial, marital and other worries play a part. So, too, are many injuries to nerves, even by invisible but poisonous coal tar gas. (3) Aggravations of pre-existing diseases or defects are as compensable, in most states, as an original or new injury. (4) Employers take workmen "as is," this is, without any warranty as to any previous state of health, known or unknown. Hence effects of injuries, intensified by pre-existing, underlying or latent disease, are very often compensable. To help cripples obtain jobs and to assist in the payment of such aggravations, the majority of states now have second-injury or similar funds. (5) Where a man sustains a nervous shock, producing physiological injury, not a mere emotional impulse, he meets with an accident. Physical impact, while common-law cases, is not necessary in compensation cases. If during a fright he falls and injures himself, or "drops dead," the injury is by accident.

Under Incapacity it is stated that one who has only a partial physical disability may obtain total incapacity payments on proof that he was unable to market his remaining capacity for work. And for permanent, total disability the disability must be permanent, that is, one that will continue for life or indefinitely. The fact that it is merely conceivable, but not probable, that improvement will take place, does not prevent a finding that the disability is permanent.

Throughout the volume there are numerous pertinent citations of court decisions and there is an excellent index and a bibliography.

Except for certain medical aspects this book is more suitable for a legal review. But it is a rare combination of research and synthesis, a work to be deeply pondered. It offers no short cuts or magic solutions but it contains the clearest discussion of its subject matter that I have ever encountered. Its use should bring about more accord in controversial or litigated issues.

C. P. MAY, M. D.

The Venous Pulse and Its Graphic Recording: By Franz M. Groedel, M. D. New York, Brooklyn Medical Press, 1946. Pp. 216. Price $5.50.

The author and his colleagues have been interested in the study of the venous pulse for many years. This small monograph presents some of their methods and results as well as review of the pertinent literature. Dr. Groedel correctly emphasizes the point of view that we cannot complete our knowledge of the circulation until we study the venous blood stream with the same interest and enthusiasm as we do the arterial blood stream. The author candidly admits that the study of the venous side of the circulation is difficult because of the presence of many variables. The development of modern methods of recording, however, has made it desirable to attack the problem again.

The monograph is divided into three sections. The major part is devoted to a detailed discussion of the phlebogram, the graphic representation of the venous pulse. The other two sections are concerned with the pneumo-cardiogram and the esophago-cardiogram, respectively.

The author uses an oscillograph and galvanometer system similar to that found in the electrocardiograph in conjunction with a high frequency microphone. Two types of waves are distinguished in the phlebogram: (1) the venous tidal waves (a, c, v, etc.) reflect the pressure-suction effect of the heart upon the blood flow in the larger veins and are therefore primarily manifestations of fluctuations in intrathoracic pressure; (2) the concussion waves due to the concussion of the blood column in the large veins caused by the opening and closing and contraction of the heart chambers. Although this method of analysis represents a distinct advance, it is apparent that much more work needs to be done to determine the origin and significance of the various waves found before the method can be used in diagnosis.

The author uses a gas mask connected to the microphone for the determination of the pneumo-cardiogram. Likewise, an esophago-cardiogram is obtained by connecting an Einhorn cardia-dilator with the microphone. These records are correlated with the phlebogram. Such a comparison indicates that when proper technics are used, the three types of records represent nearly identical tracings reflecting the mechanical events that are taking place in the heart and the intrathoracic pressure changes due to systole and diastole of the various heart chambers and to the inflow and outflow of blood from the thorax.

This monograph merits the serious consideration of those who are interested in the circulatory system. Phlebography was enthusiastically taken up after the first World War and almost as enthusiastically dropped when it became apparent that technical difficulties made it difficult to obtain good phlebograms. The strides made in electrocardiographic diagnosis created the feeling that most of the information given by the phlebogram could be obtained much more simply and clearly with the electrocardiogram. While this is true of many clinical conditions, it is now apparent that other methods must often be used to supplement the study of the electrocardiogram if we are to diagnose successfully various clinical conditions. Phlebography should prove of great value in this connection. The methods are sufficiently developed at the present time and enough is known of the significance of the various waves to justify its use in clinical examination.

H. S. MAYERSON, Ph. D.

Medicine in Industry: By Bernhard J. Stern, Ph. D. New York: The Commonwealth Fund, 1946. Pp. 209, tables 15. Price $1.50.

This small book is another of the several studies of the New York Academy of Medicine Committee on Medicine and the Changing Order.

In this monograph, Dr. Stern, Columbia and Yale University sociologist, surveys the past accomplishments in industrial medicine and draws from the data suggestions for future development. The data used in the fourteen tables are derived from dependable sources, as from the U. S. Department of Commerce Bureau of the Census, the U. S. Department of Labor, the U. S. Public Health Service, Senate Subcommittee Reports of the War Manpower Commission, and so forth. Certain references used in the body of the text, however, do not appear to have been so carefully chosen. For example, on page 162, the author contents himself with 1939 source material to arrive at such deductions as the frequency of laboratory examinations as a part of the physical examination given by industrial physicians. Are these findings representative of 1946, some seven years later, when organized labor, with better understanding of the purposes of industrial medicine, complains less about factual laboratory data?

Probably the best written of the seven chapters of the book is Chapter II, Social and Legislative Backgrounds. This chapter, with Chapter VII, Preventive Services, offers good material for reading assignment to medical students during their work in industrial medicine and hygiene.

RALPH H. HEEREN, M. D.

Group Psychotherapy: *Theory and Practice*: By J. W. Klapman. New York, Grune & Stratton, 1946. Pp. 344. Price, $4.00.

Group psychotherapy has been practiced, after a fashion, since the advent of mankind. But, as a selfconscious discipline with an attempted appraisal of causes and effects group psychotherapy is of relatively recent origin.

Its concepts, theories and technics are still in an incipient stage, but recent, urgent stresses in world affairs have served to accelerate and intensify the need for it. Because of increasing numbers of military and civilian psychiatric casualties and enlarged demands for psychiatric services in general, group-psychotherapy has suddenly been called upon to fill a role of unprecedented proportions.

While this art is comparatively new there has been time and opportunity for certain exact formulations. It is time for a summation of current usages to indicate some guiding, even though tentative, principles for those who contemplate employing group-psychotherapy.

It is believed that the important contribution group-therapy makes to the healing arts constitutes more nearly a living-through process in relation to the social setting than any other form of psychotherapy.

The range and variety of groups which may be treated by group-psychotherapy are seemingly endless. If it is borne in mind that the principles and dynamics of group-psychotherapy are essentially the same for all groups, though the modes of application may vary, few difficulties need arise in attempts to treat new, untried groups.

Group-psychotherapy is more than a make-shift in the service of economy. It extends the service of each practitioner to more patients than can be individually treated. It has the unique advantage of being able to utilize the collective will, to mobilize the contagion of the group, for therapeutic gains. It demonstrates that many persons in need of treatment, who are withholding during individual interviews, will force and discuss their psychic difficulties and traumas when they find they are not alone in their troubles. The profound importance and value of this excellent publication goes without saying.

C. P. MAY, M. D.

Suggestion and Hypnosis Made Practical: By Samuel Kahn, M. D., Ph. D. Boston, Meador Publishing Co., 1945. Pp. 200. Price, $3.00.

Suggestion is the most powerful force man uses on man. Success in human relationships is certain for those who understand and use it. It is universal in its influence and is a determining factor in getting what one wants.

The therapeutic value of suggestion in the treatment of disease has been known to mankind since the earliest stages of recorded history and it was through the efforts of Mesmer, about 1770, that a real beginning was made in what is now known as scientific psychotherapy.

Transference of ideas from one person to another is cosmic; it is this that makes possible the great effectiveness of suggestion. Ideas are contagious because of the mechanism of suggestion. Thoughts — whether they are noble or evil — if repeated often enough and with conviction, become forces that influence actions.

Generally, a patient prefers to have his problems solved by the doctor. In suggestion and hypnosis, the operator does the thinking. In analysis, the patient thinks for himself and solves his own problems under the physician's guidance.

Hypnosis, alone, cannot cure. It affords the patient no insight into the darkness of his mind, and at best can offer him only an escape from reality. Suggestion and hypnosis may undermine the individuality of the patient and make him dependent on the operator. Analysis, on the other hand, will restore his individuality, giving him strength and courage through understanding and knowledge. It is only when the patient recognizes his

internal adversary that he is able to cope with it. What has been a hidden conflict with the inner enemy becomes an open struggle. In no other way can a lasting cure be achieved. I see eye to eye with the author in these views.

Chapter 4, Auto-Suggestion, is interesting and valuable. Auto-suggestion, the use of suggestion on oneself, is a form of self-education, because the individual uses it to influence himself. With it, he talks himself into believing ideas in order to achieve specific goals. Auto suggestion is a technic for educating, training, and influencing oneself.

Powerful results can be built up by auto-suggestion. There is a "summation of results," or "habit formation." The prevention of mental conflict by re-education and autognosis, and the avoiding of negative auto-suggestion, can be successfully accomplished through repeated suggestion on oneself.

While this book treats suggestion in general there are in it chapters on special phases of using suggestion as in medicine, business, rumor-propaganda. Its use calls for realistic, inductive and creative thinking. Its author has had broad training and experience. It can help any intelligent person attain greater mental efficiency and a fuller richer life.

C. P. MAY, M. D.

Modern Clinical Syphilology: By John H. Stokes, M. D., Herman Beerman, M. D., Sc. D. (Med.) and Norman R. Ingraham, Jr., M. D. 3d ed. reset. Philadelphia, W. B. Saunders Co. 1944. Pp. 1332. Price, $10.00.

When one undertakes the task of reviewing the third edition of Stokes' Modern Clinical Syphilology he soon discovers that he is confronted with a task which challenges his ability to concentrate on the problem of syphilis, with the many ramifications of the disease. This is not a criticism of the material in the book since indeed Stokes' Modern Clinical Syphilology is an encyclopedia of syphilis. The phrase "You can look it up in Stokes" is as true of this edition as it was of former ones. The serious student of syphilis can benefit especially from referring to this volume. On the other hand, the busy practitioner who might want to look up a specific problem, such as the exact definition of Grade 1 spinal fluid, would not readily find the answer to his inquiry because he would be inclined to seek information in the chapter on Central Nervous System Syphilis, rather than in the chapter dealing with diagnostic procedures. There seems to be no clear-cut advice to the practitioner concerning the advisability of continuing to treat sero-resistant cases, and what may be expected from further treatment.

In Figure 438 concerning the prevention and the treatment of relapse, this statement is made: "Turn to 606 if 914 has failed, or use it from the start." There are other bits of advice in this figure which leads one to believe that this segment of the book has not been recently revised to consider such therapeutic agents as mapharsen or one of the other newer phenarsine preparations.

In the section on differential diagnosis between secondary syphilis and other skin ailments, one wonders why Stokes has not specified finding treponema pallidum by means of darkfield examiation of syphilitic lesions as *the* diagnostic distinction between papular or annular manifestations of syphilis and the other skin diseases being considered. Only in Figure 314 is the finding of T. P. mentioned as a diagnostic feature (as distinguished from lesions of leprosy).

The last two chapters are timely and as up to date as could be managed. It is surprising to find the relationship between military and public health syphilis handled as completely as it is in chapter 33, when one remembers that it must have been added to the book late in its production. But the ultimate in getting material to press is achieved in the last chapter in which Stokes discusses penicillin and gives a preview of its use in the treatment of syphilis. If some of his statements should not prove to be entirely accurate, one should ask the question "Could anyone have judged better except in retrospection?".

On the whole this revised edition of Stokes is a most valuable book, perhaps the world's outstanding reference book of syphilology.

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PUBLICATIONS RECEIVED

Blakiston Company, Philadelphia: Diseases of the Retina, by Herman Elwyn, M.D.

Lea & Febiger, Philadelphia: Renal Diseases, by E. T. Bell, M.D.

MacMillan Company, New York: The Venereal Diseases, by James Marshall, M.B., B.S., M.R.C.S., L.R.C.P.

C. V. Mosby Company, St. Louis: Diabetes, by Henry J. John, M.A., M.D., F.A.C.P.

W. B. Saunders Company, Philadelphia: Women in Industry, by Anna M. Baetjer, Sc.D. Peripheral Vascular Diseases, by Edgar V. Allen, B.S., M.S., M.D., M.S., F.A.C.P., Nelson W. Barker, B. A., M.D., M.S., F.A.C.P. and Edgar A. Hines, Jr., M.D., B.S., M.A., F.A.C.P

Charles C. Thomas, Springfield, Ill.: The Modern Treatment of Diabetes Mellitus, by William S. Collens, B.S., M.D., and Louis C. Boas, A.B., M.D. Peptic Ulcer, by I. W. Held, M.D., F.A.C.P., and A. Allen Goldbloom, M.D., F.A.C.P.

Year Book Publishers, Inc., Chicago: Anesthesia in General Practice, by Stuart C. Cullen, M.D.